ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 55 MARCH, 1946 No. 1

I

THE RELATIONSHIP OF DENTAL MALOCCLUSION TO VACUUM-OTITIS MEDIA AND THE USE OF DENTAL SPLINTS DURING DESCENT FROM ALTITUDES

HOWARD R. BIERMAN
COMMANDER, MEDICAL CORPS,
UNITED STATES NAVAL RESERVE
BETHESDA, MD.
AND
I. WILLIAM BRICKMAN
LIEUTENANT, DENTAL CORPS
UNITED STATES NAVAL RESERVE
PENSACOLA, FLA.

During World War II, observations in the low pressure chambers at the Naval Air Station, Pensacola, Florida, showed that from 10 to 15 per cent of more than 15,000 men developed ear block during recompression. Ear block may be defined as discomfort in or about the ear of sufficient degree during recompression to require halting any further increase of pressure.[1] Otoscopic examination in most cases confirmed the complaint of ear block, but the picture varied markedly as to degree, and occasionally no changes from the

The opinions expressed herein are those of the authors and not necessarily those of the Navy Department.

TABLE 1

METHODS USED FOR EQUALIZING MIDDLE EAR PRESSURES, BY CADETS

METHOD	TOTAL NUMBER	NO EAR DIFFICULTY NUMBER	NO EAR DIFFICULTY PERCENT	EAR BLOCK NUMBER	EAR BLOCK PERCENT
MODIFIED VALSALVA	333	297	34	36	10.8
SWALLOWING	323	296	33	27	8.4
MOVEMENT OF MANDIBLE	120	116	13	4	3.3
DEGLUTITION AND MOVEMENT OF MANDIBLE	56	52	5.9	4	7.1
MODIFIED VALSALVA PLUS MOVEMENT OF MANDIBLE	41	36	4	5	12.2
TOTAL	873	797	--	76	8.7%

TABLE 2

RELATIONSHIP OF EAR BLOCK TO DENTAL OCCLUSION IN 821 CASES

TYPE OF OCCLUSION	NUMBER OF CASES	PERCENT	EAR BLOCK NUMBER	EAR BLOCK PERCENT	NO EAR DIFFICULTY NUMBER	NO EAR DIFFICULTY PERCENT
NORMAL	447	54.4	26	5.8	421	94.2
MALOCCLUSION	374	45.6	89	23.7	285	76.3
OVERBITE	242	29.4	56	23.1	186	76.9
RETRUSION	62	7.5	18	29.0	44	71.0
PROTRUSION	70	8.5	15	21.4	55	78.6
TOTAL CASES	821	--	115	14%	706	86%

normal tympanic membrane were found. The degree of pain and its location in ear block had been thought to indicate the severity of damage within the middle ear. The otoscopic findings of this survey made such an hypothesis untenable. It was thought advisable, therefore, to study the mechanism and the prophylaxis of ear block. These studies were completed during 1942 and 1943.[1, 2, 3] The data is now presented as unclassified literature.

A survey of the most common methods used for ventilating the middle ear was made among 873 cadets undergoing "flights" in the chamber. The occurence of ear pain during descent was correlated with the method used (Table 1.) Thirty-four per cent of the group used a modified Valsalva method, 33 per cent used the swallowing mechanism, and 13 per cent used movement of the mandible. The remaining 9.9 per cent used combinations of these. Ear block occurred least frequently when movement of the mandible was used. The finding that movement of the mandible was the most effective means of ventilating the middle ear led to an investigation of the anatomical positioning of the mandible. The state of dental occlusion in 821 subjects was determined prior to a flight in the chamber and correlated with the ear difficulty encountered during descent.

Ear block during low pressure chamber descents was found to occur about five times as frequently in individuals with malocclusion as in individuals with normal occlusion (Table 2).

Another group of 439 aviation cadets and student officers were examined otoscopically shortly after their "flights" in the chamber. The findings were graded as follows:*

Grade I: Vascular injection of the tympanic membrane.

Grade II: Marked diffuse redness of the tympanic membrane in addition to vascular injection.

Grade III: Diffuse redness of the tympanic membrane with clear fluid in the middle ear behind the membrane.

Grade IV: Diffuse redness of the tympanic membrane with serosanguineous fluid within the middle ear accompanied by retraction of the tympanic membrane.

These otoscopic findings were correlated with the state of dental occlusion (Table 3).

*Only grades II, III, or IV were considered evidences of an acute vacuum-otitis media in this study.

TABLE 3.—COMPARISON BETWEEN NORMAL AND MALOCCLUSION CASES WITH OTOSCOPIC FINDINGS OF AERO-OTITIS MEDIA IN 439 SUBJECTS ON DESCENT IN THE LOW PRESSURE CHAMBER.

	NORMAL OCCLUSION		MALOCCLUSION	
	NO.	%	NO.	%
Negative otoscopic findings	241	88	117	70
Positive otoscopic findings	32	12	49	30
Total	273		166	
Complained of ear block pain	35	13	58	35

Only 12 per cent of those individuals with normal occlusion, exhibited otoscopic changes sufficient to warrant the diagnosis of an acute aero-otitis media. The remaining 88 per cent showed some scattered, vascular injection of the tympanic membrane usually with retraction, but without abnormal otoscopic findings.

Conversely, a full-blown picture of acute aero-otitis media was frequently discovered by inspection, but upon questioning no history of ear pain could be elicited. This situation occurred equally in cases of normal occlusion or malocclusion. Hearing in the affected ear was usually impaired in all cases of Grade II, III, or IV. In most cases the nasal resonance of the voice, common in ear block, was recognized by the subjects.

The fact that ear block occurs more frequently in individuals with faulty occlusion than in persons with normal occlusion suggested that repositioning the mandible might have a beneficial effect in those individuals with malocclusion. This was supported by the finding that voluntary repositioning of the jaw (yawning) proved to be the most effective method in middle ear ventilation on descents.

Horseshoe-shaped dental splints were made of acrylic resin (Fig. 1). The intermaxillary distance was increased 8 mm. along the biting surfaces and the width was 12 mm. The splint was retained in the mouth during the entire period of descent. The rate of descent was approximately 5,000 feet per minute. The splints were inserted at 30,000 feet. The incidence of ear block occurring on these descents is compared with the occlusal findings and descents without the splints (Table 4).

The incidence of ear block occurring on descents during which every man used a dental splint, is 5.2 per cent of those individuals with normal occlusion, and 9.1 per cent of those individuals with

COMPARISON OF INCIDENCE OF EAR BLOCK WITH AND WITHOUT DENTAL SPLINTS

	WITHOUT SPLINT EAR BLOCK		WITH SPLINT EAR BLOCK	
	NO. OF CASES	PERCENT	NO. OF CASES	PERCENT
NORMAL	26	5.8	11	5.2
MALOCCLUSION	89	23.7	27	9.1
OVERBITE	56	23.1	22	11.1
RETRUSION	18	29.0	4	8.1
PROTRUSION	15	21.4	1	3.8
TOTAL	115/821-14		38/502-8.1	

TABLE 4.

malocclusion experienced ear block. The dental splints significantly reduced the incidence of ear block only in individuals with malocclusion.

DISCUSSION

Manipulation of prepared sections cut through the auditory orifice and the mandibular condyle show that, on closure of the bite, the wrinkling of the external pterygoid muscle is sufficient to compress the membranous part of the auditory tube.* Overclosure or malposition of the jaw enhances this compression and also shortens the span of the tensor veli palatini muscle. This loss of span of the tensor veli palatini muscle from its origin at the membranous part of the auditory tube to its insertion in the soft palate impairs the efficiency of this muscle in opening the auditory tube during swallowing. Thus in malposition of the jaw, there is not only compression of the auditory tube by the external pterygoid, but the usual efficiency of the mechanism of opening the tube by swallowing is impaired.

The simplest method for ventilating the middle ear is swallowing. In certain individuals, however, swallowing is ineffective and the individual requires the aid of other maneuvers or a combination

*Sections prepared by Dr. J. B. Costen of St. Louis, Missouri.

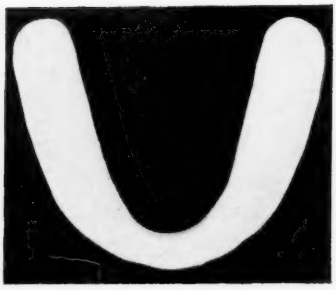


Fig. 1.—Dental splint made of acrylic resin for use in reducing the incidence of ear block in low pressure chamber descents.

of maneuvers to obtain satisfactory middle ear ventilation. Excessive mandibular movement, on the other hand, although most effective changes the normal contour of the face thus altering the fit of an oxygen mask and increasing the opportunity for leakage of atmospheric air. An attempt to combine deglutition with a simultaneous downward and forward movement of the jaw has proven a difficult maneuver to learn for mass instruction.

Ventilation of the middle ear for passengers can be readily and safely obtained without the modified Valsalva maneuver. Merely swallowing with the nares held closed is sufficient to allow air to pass into the middle ear.

Costen,[4] Wilhelmy,[5] and Lowry[6] have demonstrated that repositioning the mandible of individuals with overbite or loss of interdental space, due primarily to loss of teeth, was effective. The use of dental splints in cases of malocclusion with the normal complement of teeth to reposition the mandible proved the effectiveness of such an appliance in ventilating the middle ear. The effectiveness of the dental appliance appears to be mainly in increasing the intermaxillary distance. The increase in salivation caused by the splint stimulates swallowing which undoubtedly assists in the ventilation of the middle ear.

The splint used in the early studies interfered prohibitively with speech. Further advances in the development of the splint permitted self-retention, avoided interference with articulation, and maintained its effectiveness (Fig. 2). This device also has been developed to fit

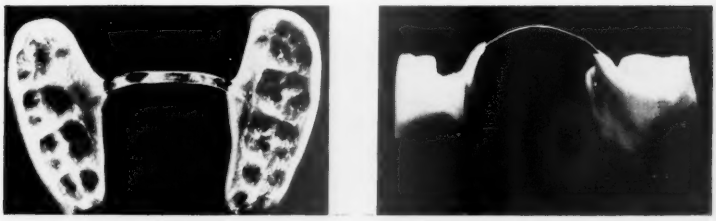


Fig. 2.—Dental splints constructed with a lingual bar to permit speaking and self-retention in the mouth for pilots and aviation personnel in aircraft.

all types of occlusion and is suitable for passenger use in transport aircraft.

SUMMARY

1. Pain in the middle ear during descent in the chamber may occur without marked changes in the tympanic membrane.

2. A survey of methods to ventilate the middle ear revealed that a modified Valsalva and swallowing maneuvers were the most common methods in use in the low pressure chamber. The most effective method, however, was movement of the mandible. Pain in or about the ear during increase in ambient pressure is not a reliable indication of aero-otitis media without confirmation by an otoscopic examination.

3. Ear block on descent in the low pressure chamber occurs about three to five times more frequently in individuals with malocclusion than in those with normal occlusion.

4. The incidence of ear block in cases of malocclusion was reduced materially when dental splints were used.

CONCLUSION

Movement of the mandible is the most effective method of maintaining ventilation of the middle ear on descent in the low pressure chamber. Ear block on descent in the low pressure chamber occurs about three to five times more frequently in individuals with malocclusion than in those with normal occlusion. The use of dental

splints on descents in the low pressure chamber materially aided ventilation of the middle ear in individuals with malocclusion.

NAVAL MEDICAL RESEARCH INSTITUTE
NATIONAL NAVAL MEDICAL CENTER
BETHESDA, MARYLAND.

CARMI, ILLINOIS.

REFERENCES

1. Brickman, I. W., and Bierman, H. R.: Relationship of Dental Malocclusion to Ear Block in the Low Pressure Chamber, Physiological Research Section, Naval Air Training Center, Pensacola, Florida, March 1943.

2. Brickman, I. W., and Bierman, H. R.: Prevention of Ear Block by the Use of Dental Splints, Physiological Research Section, School of Aviation Medicine, Naval Air Training Center, Pensacola, Florida, May, 1943.

3. Brickman, I. W., and Bierman, H. R.: The Syndrome of Ear Block, Aero-Otitis Media, and Malocclusion, Physiological Research Section, School of Aviation Medicine, Naval Air Training Center, Pensacola, Florida, June, 1943.

4. Costen, J. B.: A Syndrome of Ear and Sinus Symptoms Dependent Upon Disturbed Function of the Temporo-mandibular Joint, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 43:1 (March) 1934.

5. Wilhelmy, G. E.: Ear Symptoms Incidental to Sudden Altitude Changes and the Factor of Overclosure of the Mandible, U. S. Naval Med. Bull. 37:533-341, 1936.

6. Lowry, R. A.: Loss of Intermaxillary Distance: Effect on Aviators and Relief by Interdental Splint, U. S. Naval Med. Bull. 37:367-380, 1939.

II

DENTAL TREATMENT FOR THE PREVENTION OF AEROTITIS MEDIA

LIEUT. COMDR. WILLIAM J. KELLY

DENTAL CORPS, UNITED STATES NAVAL RESERVE

AND

HENRY W. LANGHEINZ, D.D.S.

The dental measures here reported have been successful in the prevention of recurrence of aerotitis media in 46 out of 50 candidates for submarine service whose ears blocked previously when submitting to pressure changes. The procedures used and a discussion of the possible reasons for success are presented.

Aerotitis media is the condition that results from a difference between air pressure in the middle ear and that in the surrounding atmosphere. It is the difference between the two pressures, not the absolute value of either pressure, that calls forth the series of tissue changes known collectively as aerotitis media. Equalization of pressure normally occurs by the passage of air through the eustachian tube during its momentary opening as in the act of swallowing or of yawning. Inability or failure to open the tube voluntarily at frequent intervals during a rapid change in pressure of the surrounding atmosphere is thus the inciting cause of the pressure differential that is the immediate cause of the lesions of aerotitis media. All efforts to prevent aerotitis media must therefore be directed toward maintenance, or restoration, of normal functioning of the eustachian tube.

Previous efforts in dental treatment for the prevention of aerotitis media have been based on the concept that a decrease in mandibular height (a "closed bite") interferes with normal functioning of the eustachian tube. In 1934, Costen[1] reported observations on patients who had impaired hearing associated with a closed bite; by "opening the bite" the hearing acuity of many of these patients was improved. Wilhelmy[2] promptly tried the same method of treatment with aviators who had recurrent ear symptoms incident to rapid changes of

From the Medical Research Laboratory, U. S. Submarine Base, New London, Conn.

altitude. He reported that the insertion of dental splints to "open the bite" seemed to give relief in a small group treated. A larger group (33 men) was similarly treated by Lowry, who reported in 1939 that the subjective impressions of 26 men followed up were as follows: 9 were symptom free, 14 felt some improvement from wearing the splints, and 3 derived no benefit. Harvey and Morant[4] of the Royal Air Force, however, reported that they had used splints in cases of aerotitis media and found no improvement. They also found that aerotitis media was no more prevalent among men with malocclusion than among those with normal occlusion. Teed[5], in a study of subjects at the Submarine Base, New London, Connecticut, following runs in the pressure chamber, expressed considerable doubt that overclosure of the mandible could be classed among the causes of eustachian tube failure. Another group of investigators[6] at a Naval Air Station made the observation that ear block was found to occur five times as frequently in individuals with malocclusion as in individuals with normal occlusion.

The lack of agreement in these reports induced Captain C. W. Shilling,[7] in charge of the Medical Research Laboratory, U. S. Submarine Base, New London, Connecticut, to suggest that an effort be made to achieve a clarification of these divergent views and one of the writers was ordered to conduct an investigation of the possibilities of the dental correction of the causes of aerotitis media.

The investigation was planned to determine the effects, not only of overclosure of the mandible, but also, because of certain observations the authors had previously made, of functional malocclusion upon eustachian tube function. The study was conducted at the U. S. Submarine Base, New London, Connecticut, upon candidates for the submarine service.

The rigid physical examinations and screening tests employed in examining men for submarine service at the Medical Research Laboratory excluded all candidates except young, healthy, normal males. Included in these examinations were audiometric studies of every man by the Sound Laboratory of Medical Research and a loss of more than 20 decibels in all tones below 4096 double vibrations per second was considered cause for rejection.

Thus many extraneous factors were initially eliminated and a wealth of good clinical material was available in that all candidates for submarine training are required to take 50 pounds air pressure in a recompression chamber to qualify for instruction in the use of the escape lung. This is three times the pressure to which any aviator is

possibly exposed on descent and therefore provided a thorough test of any corrective measures employed. The first step in the project was to review available articles on aviation dentistry[8] relating to aerotitis media. This review revealed a number of points common to all articles as follows: all conclusions were based upon subjective symptoms, relying on whether the man stated his ears had or had not improved following treatment; the classification of malocclusion was based upon a static relationship of the jaws; subjects were selected with overclosure of the mandible; and x-rays of the temporomandibular articulation in open and closed positions were not utilized as diagnostic aids.

With these points in mind and to rule out as many variables as possible, this investigation was accomplished as follows:

1. Those men with any interference in mandibular function who showed aerotitis media following exposure to pressure were taken at random for treatment. No effort was made to select the men on any other basis, such as amount of adenoidal tissue, history of ear disease, etc.

2. Subjective symptoms were not used as a criterion of success or failure, but only the objective observations made by an otologist, independently of this investigator. The otologist graded all ears from 0 to 5 depending upon the amount of ear damage shown otoscopically after exposure to pressure. The same classification was used both before and after dental treatment. The classification follows:

Grade 0—Normal.

Grade 1—Retraction with redness in Shrapnell's membrane and along the manubrium.

Grade 2—Retraction with redness of the entire drum.

Grade 3—Same as Grade 2, plus evidence of fluid and hemorrhage in the drum.

Grade 4—Hemotympanum, perforation of the drum, or both.

Grade 5—Drum blue black in color and tympanum filled with a serosanguineous fluid, but no air bubbles.

3. All men were examined with a nasopharyngoscope, as well as an otoscope, before and after treatment.

4. All men were given uniform instruction in the Valsalva maneuver to clear their ears before going into the chamber.

5. Successful cases were rechecked through the chamber some months later to determine what degree of permanency was afforded by the treatment.

6. Two control groups were used:
   a. An untreated group that did not have aerotitis media on the first chamber run was repressured at intervals.
   b. An untreated group that did show aerotitis media on the first chamber run was repressured at intervals.

Technique Used. The following procedures were used in the study of each patient. X-ray films of both temporomandibular articulations, using the mastoid approach from the opposite side, were taken in both the open and the closed position.

With a suitable material, impressions of both jaws were made and casts run up in stone. The casts were mounted on a Hanau articulator. This form of articulator was selected because it produces equivalent movements of the mandible of a type satisfactory for the purpose of this study.

The next step was to transfer the upper cast to the articulator with the aid of a face bow. The purpose of this was to have the upper cast in the same relation to the hinge of the articulator as the maxilla is to the temporomandibular articulation.

The mounting of the lower cast is important in that all registrations should be taken in unstrained positions for accuracy. This is necessary because the magnitude and direction of forces and their points of application during a registration are beyond the control of both the operator and the patient. It is logical to assume, therefore, that methods to record jaw relationships in which there is pressure may be unreliable. Of all the magnitudes, directions, and points of application of forces that a patient can possibly exert, there is only one reliable and definite combination, and that is, the magnitude of the force Zero, a force which has no direction or point of application.

With this in mind, the patient was placed in a relaxed, upright position, instructed to swallow, open to a rest position, and allow a thin piece of low fusing wax to be inserted between the arches, and then instructed to close gently until the first point of contact was made through the wax by any two or more opposing teeth, but no further closure was permitted. The wax was then chilled and carefully removed. This was repeated until two or more "bites" agreed.

It is realized that biting into wax requires the application of some force and in order to reduce this as much as possible, a very soft heated wax was used and the patient instructed to close gently.

The condyle paths on the articulator were then set by having the patient protrude the lower jaw and new wax registrations were made with the patient's jaw in this position and the articulator adjusted accordingly. Lateral settings on the articulator were determined by the formula $L=H/8+12$, where L is the lateral condyle inclination in degrees and H is the horizontal condyle inclination in degrees as established by the protrusive relation.*

A study was then made of this relationship on the articulator and the points of premature contact were marked and compared in the mouth. The models were then run through all paths of excursion and any point of interference noted.

These points were then checked in the mouth by having the patient duplicate the movements with carbon paper between the teeth. The inclined planes of the teeth around the point of premature contact were studied on the models and in the mouth. Then with a small stone interfering inclined planes were reduced to provide unhampered excursions.

The intermaxillary distance in static occlusion was not altered in any way, since the cuspal points or occlusal sulci were not touched. Only planes which offered resistance to free excursion were reduced. Any cusps that were removed did not initially assist in maintaining the intermaxillary distance. Missing teeth were replaced with suitable fixed or removable bridgework; the occlusal portion of tilted teeth was placed in contact with the opposing arch by means of gold inlays and unsatisfactory fillings were replaced.

The object of this procedure in each case was to restore an occlusal plane, fully equilibrated, that permitted unhampered movements of the mandible in the various excursions and to provide adequate support in all balancing and working positions.

All cases were carefully studied for possible overclosure or loss of intermaxillary distance. The following factors were taken into account.

(1) The appearance of the patient;

(2) Possible superior displacement of the condyle heads in the fossae as revealed by the x-ray films;

(3) Gnathodynametric readings[18] to locate that occlusal relationship (intermaxillary distance) which allowed the maximum biting force to be registered, less the normal free way space;

*A full explanation of the operation of this formula is contained in the instruction book[9] accompanying each Hanau articulator.

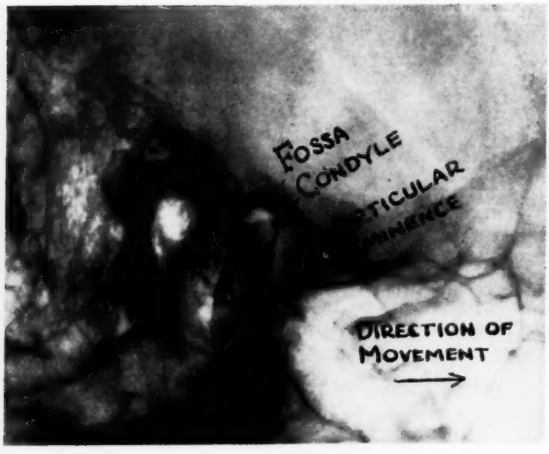


Fig. 1.—Right side, closed position before treatment, the condyle head is superiorly displaced in the glenoid fossa.

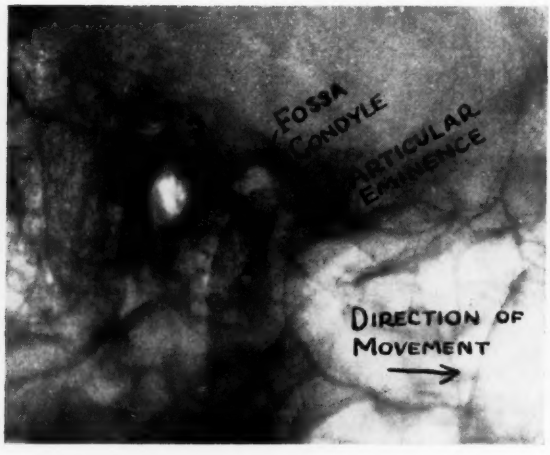


Fig. 2.—Right side, closed position after the insertion of a dental splint. Note how the condyle head has begun to assume a lower and more normal position in the fossa.

(4) Missing posterior teeth which often pointed to a possible loss of this intermaxillary distance.

The previous dental approach to this problem has dealt almost exclusively with the insertion of splints to "open the bite." We feel that the widely divergent views as to the efficacy of this procedure and the poor degree of success, or lack of it, in the hands of some investigators may be attributed to the fact that splints were used in cases where they were not indicated and by over-opening increased strained articular relations, thus further aggravating an already abnormal situation. We believe that the use of splints is a definite adjunct to the dental treatment of aerotitis media in some cases. Splints should only be employed, however, where indicated and great care must be exercised not to open beyond normal limits and to maintain a correct free way space between the upper and lower teeth with the jaws at rest.

Over-opening of this space due to tilting and drifting of the teeth or other causes was carefully watched for.

Results. Over a period of 10 months, 50 cases of aerotitis media were treated using the foregoing procedures. Following dental treatment the men were again submitted to 50 pounds pressure and when they emerged from the chamber their ears were examined otoscopically by the Medical Research otologist. Any man showing a Grade 1 ear, either unilaterally or bilaterally, was regarded as an unsuccessful case.

This is a very high standard to meet as a Grade 1 ear shows only a pinkness around the periphery of the drum and along the handle of the malleus and a man can give himself this type of ear while under pressure when executing the Valsalva maneuver if he has the slightest degree of eustachian tube blockage. Forty-six of the 50 men, however, came through pressure after treatment with absolutely negative or Grade 0 ears, whereas before treatment, they had had from Grade 3 to Grade 5 ears either unilaterally or bilaterally.

In these 46 successful cases the conclusions were based upon objective findings, that is, otoscopic examination, which is the only reliable criterion of the success or failure of any treatment for aerotitis media. We found that little reliance could be placed upon subjective symptoms because many men took 50 pounds pressure and on leaving the pressure chamber stated that their ears felt normal and that they had no pain or discomfort; even when otoscopic examination showed that they had Grade 3, 4, or 5 ears. This could be parti-

Fig. 3.—Right side, open position before treatment. The condyle is unable to leave the fossa and is impacted against the anterior wall of the fossa. This position possibly accounted for a recurrent otalgia complained of by the patient.

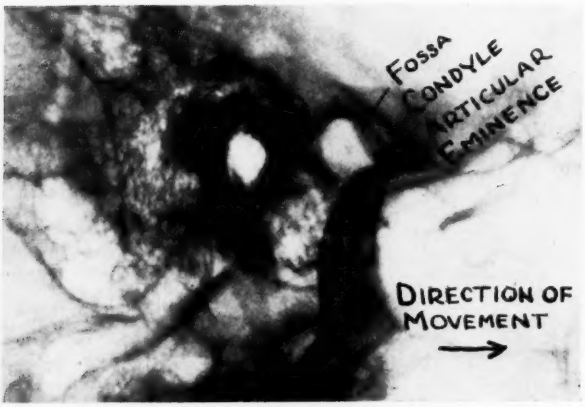


Fig. 4.—Right side, open position after the insertion of the splints. The condyle now is able to make an excursion and assume a position below the articular eminence.

ally accounted for by the universally high motivation among the candidates and fear of disqualification.

In this series of 46 cases we found five in which there was an excessive intermaxillary opening caused by the early loss of the 6-year molars and the subsequent tilting and drifting of other teeth in the molar region. These five men were treated by the reduction of the occlusal surfaces of the offending tooth or teeth or by the removal of third molars so that a more nearly normal vertical dimension was restored; these procedures, plus removal of lateral premature contacts, sufficed to give a successful result. These five cases were of great interest to us as the possibility of exaggerating the intermaxillary distance had never previously been considered as a possible factor in aerotitis media. Following any other concept but a functional one this factor would have been ignored and we believe that this accounts for failures where splints are used indiscriminately.

In only three of the 46 cases were spints employed. In each of these a number of molar teeth were missing and superior displacement of the condyle heads could be demonstrated radiographically in the glenoid fossae. In each case postoperative x-ray films were taken as a check. The splints were so constructed that the resulting occlusal plane was in harmony with the mechanical and physiological requirements of the temporomandibular articulation thus permitting unhampered excursions of the mandible.

Figures 1-8 show the x-rays of one of the three men and illustrate the importance of properly projected radiographs in the diagnosis and treatment of these conditions.

In this group of 50 men all types of adenoids were found, ranging from enormous to small. Twenty of the patients had enormous adenoids that covered the ostia of the tubes. We have failed so far to find any correlation between the type of lymphoid tissue found in the nasopharynx and the success or failure of a dental treatment that follows these methods of diagnosis and treatment.

The mouths of ten of the men in this series judged according to the standard concept of static occlusion would be classed as normal, but when studied in motion, pronounced premature and interfering contacts could be demonstrated causing the condyle heads to assume strained articular relations, brought about in many instances by aberrant inclined planes on large occlusal inlays and amalgam restoration. Reduction of the inclined planes to provide unhampered movement in all excursions was sufficient treatment.

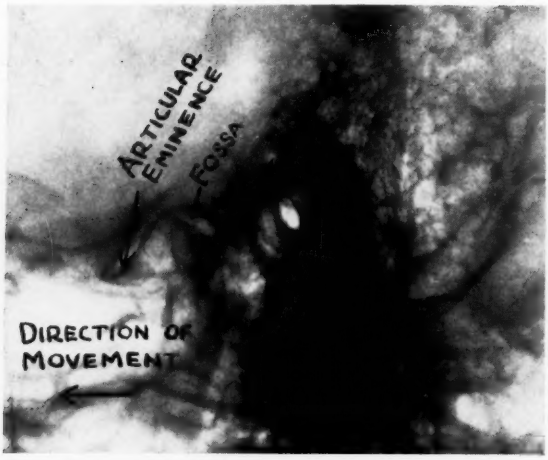


Fig. 5.—Left side, closed position before treatment. The condyle head has a superior displacement in the fossa.

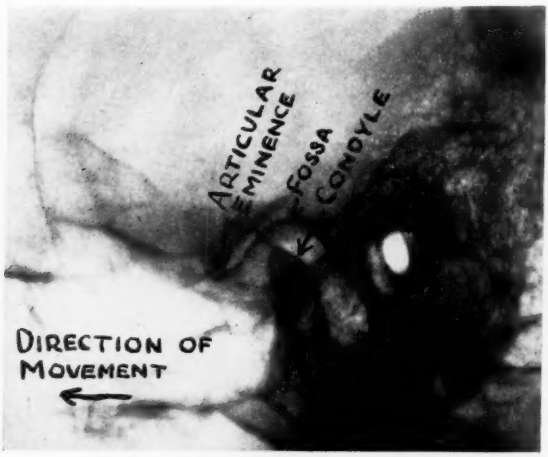


Fig. 6.—Left side, closed position after the insertion of the splint. The condyle head is now lower and in a more normal position in the fossa.

It was further noted in this series that where there was unilateral aerotitis media the occlusal interference in lateral excursion was always on the side away from the affected ear, while in lateral movement on the side away from the normal ear there was little interference. These men were treated by simply removing the lateral premature contacts on the one side.

Radium therapy was successfully used in three of the four men in whom success was not attained by means of dental treatment. Included in the series of 46 cases were two that had a number of radium treatments but still showed aerotitis media when repressured; these responded to dental treatment.

Control Groups. The untreated group that did not have aerotitis media on the initial chamber run was repressured at intervals with no significant deviation. This held true as well for the untreated group that did show aerotitis media on the initial run.

In an attempt to rule out any psychic factors in dental treatment a group of 10 men was taken who had experienced so much discomfort in the pressure chamber that they were released before reaching 50 pounds pressure. Both ears of each of these men were judged Grade 3 or Grade 4. Nothing was done to change the existing jaw relationship but the men were unaware of this; they were led to believe that a thorough dental treatment had been carried out. The group was then repressured and on leaving the chamber all showed the same type of ears as they had on the first run. The only difference was that five of the men "stuck it out" to 50 pounds. We were able to get five of the men and institute regular dental procedures and then repressure them. Examination of the ears showed them all to be negative. This led us to the conclusion that any psychic factor involved might assist a man to withstand pressure of 50 pounds, but would not affect the degree of aerotitis media.

We were able to repressure ten of the 46 men, some four months after treatment; all ears were still negative, which indicates that the effects of the treatment are permanent.

Additional Observation. Many cases were seen (not in this series) in which there was definite static or functional malocclusion or both, and the men came out of the pressure chamber with unaffected ears. All types of adenoid tissue was present. Temporomandibular x-rays of a number of these cases (25) in both open and closed positions disclosed uniformly flat fossae, presenting no mechanical problem to any sort of movement.

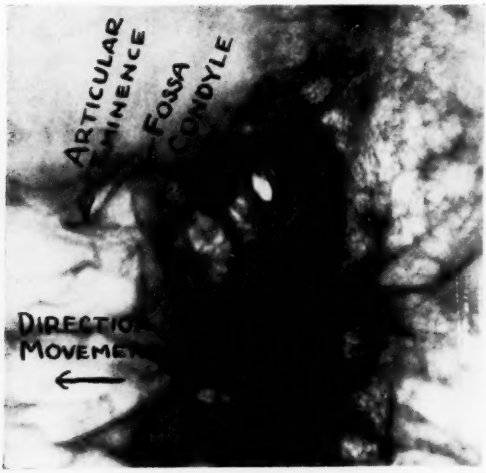


Fig. 7.—Left side, open position before treatment. The condyle head is unable to leave the fossa, possibly blocked by a traumatized meniscus or disk caused by continued occlusal disharmony.

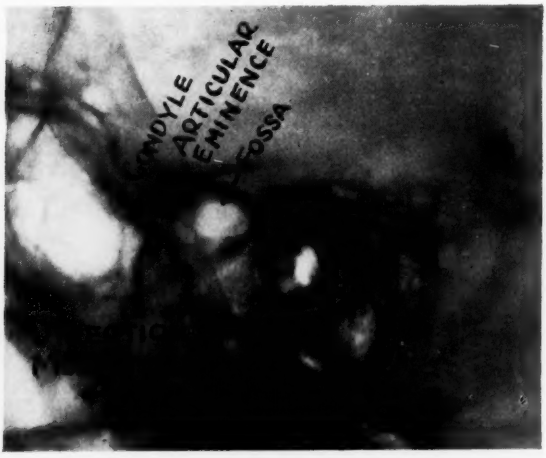


Fig. 8.—Left side, open position after the insertion of the splint. The condyle is now able to make a more normal excursion and the symptoms of otalgia have subsided.

DISCUSSION

The rationale for the methods used by Wilhelmy and Lowry was based upon the Costen hypothesis[1] which postulates that in cases of overclosure of the mandible the internal pterygoid muscle relaxes and crowds the soft tissue against the pharyngeal opening of the eustachian tube, causing a mechanical obstruction, and that the insertion of splints to restore the proper relation corrected this condition permitting the ventilation of the middle ear.

Seaver[10] believed that as the tensor palati muscle (considered the most important of the three muscles opening the tube) receives its innervation from the fifth cranial nerve, it is affected by painful stimulation of the sensory distribution of the fifth nerve, especially the mandibular division, and that this painful stimulation is present in malocclusion. He further stated "as stuffiness of the middle ear is a common complaint in mal-occlusion it is assumed that the function of the tensor palati muscle in opening the tube may be inhibited."

Neither of these theories satisfactorily explains the results obtained in the present study. The observations made are in agreement with a hypothesis suggested by Dr. Stacy Guild of Johns Hopkins, in conversation with one of the authors. The hypothesis is that free lymphatic drainage of the middle ear and the eustachian tube is interfered with by fascial and muscular tensions due to the abnormal jaw movements and that restoration of normal movements in some manner permits a free drainage of lymph, which in turn quickly relieves the mucosal congestion of the eustachian tube. Supporting this hypothesis are the following facts:

Most[11-14] states that "the lymph vessels in the middle ear connect through the lymph capillaries of the Eustachian tube with those of the lateral pharyngeal wall . . . that they pass the rear circumference of the tube, through the cartilaginous wall and travel between it and the bone to the lateral pharyngeal gland which lies in the buccopharyngeal fascia. *The lateral pharyngeal gland is the first and most important lymph node draining the eustachian tube and middle ear.*" (Authors' italics.)

Gray's Anatomy[15] shows that the superior pharyngeal constrictor muscle forms the lateral walls of the pharynx and that the lymph channels passing to the lateral pharyngeal lymph node are affected by the movements of this muscle.

Concerning the relation of this muscle to the muscles of mastication Shapiro and Truex[16] have this to say, "In working with this

specimen we were particularly impressed with the alternate tense and lax condition of the superior pharyngeal constrictor muscle as the mandible was moved from side to side. When it is recalled that this muscle is continuous with the buccinator through the pterygomandibular raphe, tenseness of the constrictor is readily understood as playing a secondary role to the pull of the buccinator during movements of the mandible."

As to the relation of muscle activity to the flow of the lymph, Starling[17] has written: "The only part of the body which has a continuous flow of lymph during rest is the alimentary canal . . . since the lymphatics are all provided with valves the effect of external pressure on them is to cause the lymph to flow in one direction only, towards the thoracic duct and great veins. *Hence we may look upon muscular activity as the greatest factor in the circulation of lymph.*" (Authors' italics.)

The anatomic and physiologic facts, together with our observations in the present study warrant the tentative conclusions that any conditions that interfere with the normal function of the buccinator would in turn affect the superior pharyngeal constrictor muscle, and by inhibiting its movements produce a congestion of the eustachian tube due to a stasis of the lymphatics caused by insufficient muscular activity, or due to a tourniquet effect as they penetrate the muscle layers. The restoration of an occlusal plane fully equilibrated restores the milking action on the lymphatics and removes the tourniquet effect due to strained muscular activity.

SUMMARY

1. Successful treatment of aerotitis media, using these dental methods of diagnosis and treatment, has been demonstrated in 46 out of 50 cases where the problem is approached on the basis of functional rather than static occlusion.

2. Because one control group with aerotitis media on their initial chamber run continued to show approximately the same degree of ear damage when repressured at intervals after a complete recovery and without any corrective treatment, we believe that the series of 50 cases reported in this paper would have continued to show aerotitis media on repeated chamber runs if no dental treatment had been instituted.

3. We believe we have shown that the elimination of functional restrictions of the buccinator muscle, and hence the superior pharyn-

geal constrictor due to their interrelation through the pterygo-mandibular raphe, by dental equilibration and restoration of a normal intermaxillary distance re-establishes the "milking" action on the lymphatics and removes any tourniquet effect. This stimulation of their drainage reduces edema and congestion of the eustachian tube and the middle ear.

4. We also believe that further substantiation of the theory of lymphatic stasis is borne out by the fact that the amount of lymphoid tissue present in the nasopharynx had little bearing on the success or failure of this method of dental treatment.

5. In cases where the glenoid fossae are flat, there appears to be little mechanical interference with mandibular motion, despite the type of malocclusion present, either functional or static, with slight evidence of lymphatic congestion shown.

Grateful acknowledgement is made to Captain C. W. Shilling, (MC), USN, for his advice and cooperation in making this study possible; to Dr. Stacy Guild for his interest in the investigation and for his suggestions in the course of preparing this report; and to Lt. (jg) Henry L. Haines, whose otological knowledge and skill was placed freely at our disposal and was of great assistance.

29 Craig Place.
Cranford, N. J.

REFERENCES

1. Costen, J. B.: A Syndrome of Ear and Sinus Symptoms Dependent upon Disturbed Function of the Temporomandibular Joint, Annals of Otology, Rhinology and Laryngology 43:1 (March) 1934.

2. Wilhelmy, G. E.: Ear Symptoms Incidental to Sudden Altitude Changes, and the Factor of Overclosure of the Mandible, U. S. Naval Med. Bull. 34:533-541 (Oct.) 1936.

3. Lowry, R. A.: Loss of Intermaxillary Distance; Effect on Aviators and Relief by Interdental Splint, U. S. Naval Med. Bull. 37:367-380 (July) 1939.

4. Flying Personnel Research Committee, Royal Air Force, Minutes of the Twelfth meeting of the Otological Committee, 18 May 1945.

5. Teed, W. R.: Factors Producing Obstructions of the Auditory Tube in Submarine Personnel, U. S. Naval Med. Bull. 42:303-5 (Feb.) 1944.

6. Relationship of Dental Mal-occlusion to Ear Block in the Low Pressure Chamber. From the Dental Department and Physiological Research Section, U. S. Naval Air Training Center, Pensacola, Fla., 1944.

7. Shilling, C. W.: Aerotitis Media and Loss of Auditory Acuity in Submarine Escape Training, Arch. Otolaryng. 42:169-173 (Sept.) 1945.

8. Mitchell, D. F.: A Bibliography of Aviation Dentistry, AAF School of Aviation Medicine, Randolph Field, Texas, 1945.

9. Hanau, R. L.: Full Denture Prosthesis—Intraoral Technique for Hanau Articulator Model—H, 4th edition, Hanau Eng. Co., Buffalo, N. Y.

10. Seaver, E. P., Jr: Temporomandibular Joint Malocclusion and the Inner Ear, a Neuromuscular Explanation, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 46:140 (March) 1937.

11. Most, A: Ueber den Lymphapparat von Nase und Rachen, Arch. f. Anat. u. Phys., Anat. Abt. 1901, pp. 75-94.

12. Most, A.: Topographisch-anatomische und klinische Utersuchungen über den Lymphgefassapparat des ausseren und mittleren Ohres, Arch f. Ohrenh. 64:189-203, and 233-253, 1905.

13. Most, A.: Die Topographie des Lymphgefassapparates des Kopfes und des Halses in ihrer Bedeutung für die Chirurgie, Berlin, 1906.

14. Most, A., and Bartels, P.: Lymphgefasse an Kopf, Nacker und Hals, Von Bardeleben Handbuchs der Anatomie des Meuschen, Jena, Gustav Fischer, 1909, Vol. 3, Sect. 4, Ch. VIII b.

15. Grays Anatomy, 21st Edition, Philadelphia, Lea and Febiger, 1924, pp. 642-643.

16. Shapiro, H. H., and Truex, R. C.: The Temporomandibular Joint and the Auditory Function: J. A. D. A. 30:1159-60-61-62 (Aug.) 1943.

17. Starling, E. H.: Human Physiology, 4th ed., Philadelphia, Lea and Febiger, 1926, pp. 871-4.

18. Kelly, W. J., and Langheinz, H. W.: Physiological Determination of the Vertical Dimension, Dental Digest 49:115-117 (March) 1943.

III

DEEP INFECTIONS OF THE NECK WITH PARTICULAR REFERENCE TO THE ROLE OF DENTAL FOCI

C. C. Swann, M.D.

Asheville, N. C.

Dental disease of the lower jaw is one of the most frequent sources of deep infections involving the fascial planes of the neck. Secondary infection may occur without dental interference, but it most frequently follows molar extraction, mandibular injection or infection around a tooth, periapical abscess or alveolar trauma from any cause. It is remarkable that relatively so few complications follow the numerous dental procedures carried out in a constantly infected field. The prophylactic use of sulfonamides locally following extractions has decreased complications to a great extent in recent years.

Due to the fact that some external swelling frequently follows extraction, there is a tendency on the part of some men to ignore this very early sign of possible beginning deep infection. Few conditions are more dangerous to life than a rapidly spreading cellulitis of the neck. Careful and frequent observation following dental procedures on the lower jaw is of the utmost importance. Temperature records and blood examinations should be routine on all complicated cases. When consultation is desired, it should be requested early and the patient not allowed to become seriously ill before consultation and aid are sought.

A thorough knowledge of the regional anatomy involved is essential to correct diagnosis and surgical approach. Adequate descriptions of the fascial planes and spaces have been made by Barnhill,[1] Blassingame,[2] Mosher,[3] Grodinsky,[4] Iglauer[5] and textbooks on anatomy. The spaces of the neck which chiefly concern us are designated as the submental, sublingual, and submaxillary; the lateral pharyngeal (parapharyngeal or pharyngomaxillary) consisting of an anterior and posterior compartment; the parotid, retropharyngeal and pterygomaxillary. The latter was described by Shure,[6] and

---

Presented before the Southern Section Meeting of the American Laryngological Rhinological and Otological Society, Richmond, Va., Jan. 7, 1946.

Greenfield[7] reported pterygomaxillary infection following mandibular injection.

The hyoid bone and mylohyoid diaphragm serve to divide the neck into an upper and a lower compartment. The mylohyoid muscle is hung like a broad hammock from the mylohyoid ridge of the mandible to the hyoid bone and fuses superficially with the deep cervical fascia. Division into potential spaces results from deep projections of the superficial layer of the deep fascia. Each space is so limited by dense fascia and muscle attachments that much pressure must be exerted before infection extends into adjacent areas. It must be kept in mind that these anatomical subdivisions apply to potential spaces only, that is, areas containing connective tissue and occasional glands. A cavity develops only after infection creeps in and suppuration takes place.

Etiology and Pathology. Orton[8] states that dental origin is second only to infection of the pharyngeal and tonsillar regions in causing deep infections of the neck. Trout,[9] Taffel and Harvey,[10] Muckleston,[11] Leonardo,[12] and Williams[13] report dental abscesses as the chief source of the deep neck infection usually referred to as Ludwig's angina. The teeth most frequently involved are the second and third molars, with the first molar and the bicuspids less frequently at fault. Anatomic studies by Tschiassny[14] show that the roots of the second and third molars almost invariably extend to the mylohyoid ridge, and sometimes below it. The roots of the first molar teeth do not usually reach the ridge and the anterior teeth never reach it. His studies further reveal that perforation of a third molar occurs invariably on the inner wall due to the extreme thickness of the external wall of the alveolus posteriorly. There was found marked irregularity in the thickness of the inner and outer walls about the first and second molars, and perforation could occur on either side. Grodinsky[4] states that the alveolus anterior to the premolars is thinnest on the lateral side, hence rupture or extension usually occurs here and the route is over the external rim of the mandible.

McCaskey[15] says that dental caries may cause gangrene of the dental pulp, the infection spreading from there to the apex of the tooth and then to the alveolus, resulting in absorption of the cement of the tooth and of the corresponding wall of the alveolus. The strangulation resulting from the inflammation of the canal may cause osteomyelitis and perforation of the alveolus. Beck[16] states that infection of the gingival tissues, the floor of the mouth and the neck may result from dental caries involving the crown, and not necessarily the roots of the tooth. The inferior dental canal

may be the channel whereby infection is transmitted posteriorly and then down into the deeper structures. Involvement of the submaxillary space occurs first in the majority of instances and since the submaxillary gland extends around the posterior edge of the mylohyoid to the sublingual space, infection readily enters here. The proximity of the third molar to the posterior border of the mylohyoid makes it easy for an infection beginning from this tooth to spread simultaneously into both the submaxillary and sublingual spaces. Injection of anesthesia about the third molar may easily carry infectious material to the submaxillary space. Periapical abscesses may rupture internally above the mylohyoid line and involve the soft tissues leading directly into the sublingual space. One or both sublingual spaces may be involved, since they are in direct communication anteriorly.

When the submaxillary and the sublingual spaces are involved and infection is present between the intrinsic muscles of the tongue and at the base of the tongue, with sepsis and rapidly spreading cellulitis, the condition is known as Ludwig's angina. The parotid space is occasionally involved and infection here occurs by direct extension or through the posterior facial vein from the submaxillary space. The submaxillary space also has potential communication with the lateral pharyngeal space and deep phlegmon may develop here by infection spreading either from the submaxillary or from the parotid space. Infection within the carotid sheath may extend upward into the skull or downward into the mediastinum. Beck[16] states that when an infection encircles the pharynx as an extension from the lateral pharyngeal space, the retropharyngeal tissues will become involved. In such instances, he states, further infection in the prevertebral fascia is present also. Few references have been made in the literature to such extension, and probably only the most advanced and fulminating cases have this complication. These infections usually spread by continuity in the form of rapidly advancing inflammatory cellulitis, but may extend also by way of the lymphatics or by septic thrombophlebitis.

The invading organisms are mixed and many combinations of bacteria may occur. Beck[16] found in an analysis of 100 cases that the streptococcus appeared in 80 per cent of the cases and that the streptococcus hemolyticus was found in pure culture in about 40 per cent of all the infections. Alden[18] believes that Vincent's organism is the predominant offender and recovered it in 24 out of 26 cases, either in pure culture or in combination with the streptococcus, staphylococcus, or the colon bacillus. McCaskey[15] reports that occasionally the gas-producing organism is present, and I have had one such case

which ended fatally. Most authors agree that the streptococcus predominates with the spirochete of Vincent nearly always present and that all cases should be treated as mixed infections. The pathological tissue changes consist of marked edema, with the intracellular spaces filled with a serous, somewhat hemorrhagic exudate. The cellulitis may spread rapidly from one compartment to another, resulting in necrosis and abscess formation from loss of oxygen due to the intense swelling and impaired circulation. The amount of actual pus present is usually small and frequently no pus is found at operation. However, if proper drainage is afforded, there results always a large amount of serous, fibrinous, or hemorrhagic exudate.

Clinical Features. Painful swelling in the suprahyoid region of the neck following any dental procedure, trauma of the lower jaw, or in the presence of dental disease is the first sign of deep infection. The area involved is brawny or board-like and tender; only rarely is fluctuation present, and then late in the disease. The swelling usually spreads rapidly, but the course may be mild for several days and then suddenly assume an alarming character. In the beginning there is no redness of the overlying skin, but edema is present and there is no pitting on pressure.

Systemic signs of the infection depend upon the stage to which it has progressed when first seen and are usually those of an intense toxemia. When the infection appears anteriorly in the submental space, all symptoms are usually mild; trismus is absent and there is no difficulty in swallowing and none in respiration. The prognosis here is excellent in the light of present-day methods of treatment. Since there is overlapping of the fascial planes and potential communications exist between all spaces, there may ensue a rapid extension from one compartment to another. The submaxillary area, just anterior to the angle of the jaw, is the first involved in the majority of instances, as most infections descend posteriorly. A point of tenderness is found just below the jaw, anterior to the angle, and usually no solitary lymph nodes can be palpated. Trismus remains absent and prognosis is excellent when surgical treatment is instituted early. Involvement of the tissues of the floor of the mouth and the base of the tongue denotes extension into the sublingual space. Van Wagenen and Costello[19] conclude that cellulitis of the submaxillary region practically always precedes sublingual phlegmon.

When a dental abscess ruptures internally or infection spreads first downward along the lingual side of the alveolar process, immediately swelling of the floor of the mouth results. In either route of infection the mucous membrane is raised and edematous and the

tongue is swollen and pushed to the opposite side. There is pain and difficulty on attempting to open the mouth and embarrassment to swallowing. When both sublingual spaces are involved, the tongue is pushed upward and backward, narrowing and nearly filling the pharynx. The tongue is practically immobile and speech difficult to understand; later the tongue may be pushed forward also and speech becomes impossible, due to extension of the edema to the larynx. Respiration is interfered with and there is profuse drooling of saliva, due to infection in and about the salivary glands. Severe laryngeal edema, with asphyxia, is likely unless remedial measures are instituted as soon as the patient is seen.

Infection in the parotid space is often rapidly followed by involvement of the lateral pharyngeal space. Barnhill[20] reports a case in which parotid infection spread beneath the temporal fascia, resulting in hard, nonfluctuating swelling, and marked edema of the eyelids. Signs of infection in the lateral pharyngeal space differ somewhat as to whether the involvement is in the anterior or posterior compartment, or both. Involvement of the carotid sheath is possible, but unlikely without preceding infection anteriorly. Difficulty or inability to open the mouth, accompanied by medial bulging of the lateral pharyngeal wall and increasing swelling and induration of the tissues at the angle of the jaw, clearly indicates infection of the lateral pharyngeal space. Trismus is explained by the close relationship of the internal pterygoid muscle to the site of infection and it is never absent when this space is involved. Now signs of sepsis are more pronounced and the risk of complications becomes more grave.

Evidence of infection in the carotid sheath is not so characteristic as involvement of the space anterior to the styloid process. External swelling usually spreads downward and may extend to the clavicle; tenderness may be elicited along the anterior border of the sternomastoid muscle. Torticollis may be present and is toward the opposite side. Individual lymph nodes are usually not palpated but these can become infected and an abscess forms; when this occurs, there may be pitting on deep pressure over the area. Marked temperature changes, severe chills and sweats, with high blood counts and positive blood cultures are more likely to occur with carotid space infections. Beck[17] states that we should be guided more by the degree of sepsis than by local signs in making a diagnosis of carotid sheath infection. Sudden pain and tenderness in a distant joint or muscle indicates septic emboli, and embolism may occur in the retina. Early incision and drainage of the infected spaces will usually prevent fatal complications. Furstenberg[21] states that retropharyngeal space infections are rare in the adult

due to atrophy of the lymph glands in this space. When a virulent infection encircles the neck, it is possible for extension to occur from the lateral pharyngeal space. Pretracheal fascia infection is a grave sequence to a lateral pharyngeal involvement. It is characterized by severe dyspnea, extreme prostration, and a high degree of sepsis. Asphyxia is the paramount danger in pretracheal infections and this termination may not be prevented by tracheotomy.

Abscess of the pterygomaxillary space with involvement of the mandibular fossa is indicated by swelling and acute tenderness anterior to the auricle, over the temporomandibular articulation. Trismus is marked and the patient is able to open the mouth but slightly. Only 11 cases have been reported according to Shure.[6] The above findings, occurring within a few days following mandibular block anesthesia, indicate infection in this space.

Complications. The risk of secondary complication is not great when the infection is confined to the submental or submaxillary spaces, when drainage is established early. There is always the danger of phlebitis of the veins in these areas and the possibility of infection spreading along these channels. When the sublingual or lateral pharyngeal spaces are involved, respiratory obstruction is the most probable of all complications to occur. It is due either to blockage of the airway by an elevated and edematous tongue, or by spasm or edema of the larynx. This is always imminent in the late stages and complete obstruction may occur in a very short time. Phlebitis of the jugular vein occurs more often than is realized and a thrombus may form, with extension of the infection into the blood stream or into the mediastinum. Daily blood cultures should be begun early in the illness so that when infection does occur it may be detected before the infected thrombus blocks off the jugular vein. Bronchopneumonia frequently follows the spontaneous rupture of an abscess into the pharynx, as the patient is unable to rid himself of the purulent material which floods the trachea and bronchi. In neglected cases, the wall of the carotid sheath may become necrotic from pressure of the abscess and rupture of the internal jugular or carotid artery may result in fatal hemorrhage. It is possible for sudden death to occur with involvement of the vagus in severe carotid sheath infections.

Osteomyelitis of the mandible is a frequent complication arising in the presence of soft tissue infection and abscess, and since changes revealed by x-ray examination are late in appearing, it is likely to be overlooked. Meningitis, by an extension upward along the carotid sheath, occurs less often than mediastinitis, but when it does compli-

cate a fulminating infection below, it is usually fatal. Parotid abscess occurs occasionally following dental infections and the prognosis is excellent in the presence of early surgical interference.

Treatment. Prophylaxis is extremely important and may well determine the course and outcome of infection secondary to dental disease. Preliminary care by the dental surgeon should always be given in cases of advanced pyorrhea, dental abscesses, and compound fractures of the mandible which have gone unattended. Judicious waiting in the presence of acute dental abscess will in most cases obviate the risk of secondary infection. Sulfonamide preparations by mouth or penicillin for several days in advance is a wise precaution in cases of considerable infection where extractions are necessary. The local use of sulfanilamide and sulfathiazole powder, mixed, in all sockets of infected teeth extracted will do much to limit the infection to the tooth area. Sulfonamides, penicillin, or both, should be administered as an adjunct to surgery in all cases of deep neck infection. It is erroneous, and often fatal, to depend upon sulfonamides or penicillin to take adequate care of these infections. Deep x-ray therapy has been advocated along with other nonsurgical measures, but we must consider that it may increase tension in tissues already under intense pressure. The beneficial physiologic reaction is not sufficient to warrant placing much value on the roentgen ray as a curative agent.

In involvement of spaces other than the submental and submaxillary, voluntary intake of fluids is greatly reduced or absent. These may be given by nasal tube or intravenously. The patient is already extremely restless and retention enemata and hypodermoclysis tend to aggravate the restlessness. Sedatives are highly beneficial, but should be used only when the patient can be kept under constant observation by a competent nurse. Hot packs to the involved area at frequent intervals give comfort and may favor resolution. The role that penicillin will play in the effective treatment of these infections is as yet not fully determined. Gaines and Hatcher[22] report favorable results with this drug and I had one complete recovery from a large submaxillary abscess where operation was refused.

Barnhill[1] states that every deep abscess of the neck is a case for surgery and that operation should be performed as soon as it is reasonably certain that an abscess is present. He has found that abscess of the neck has a natural tendency to wall off and thus by the time the diagnosis is made, it may usually be safely assumed that it is

already well isolated. Williams[13] states that fluctuation and pus will develop in about 50 per cent of the cases and then only after a matter of days. Mosher[3] makes the statement that to wait for fluctuation is to invite secondary complications.

When an abscess is located definitely in the submental space, a midline incision in the submental triangle is made, extending from the symphysis to the hyoid bone. The gloved finger is inserted and the muscle planes gently explored. The relief of tension and the establishment of drainage are usually all that is necessary to provide prompt and rapid recovery. Occasionally an abscess will point just beneath the mucosa of the floor of the mouth, between the tongue and the medial surface of the alveolus. Free incision and gentle exploration with a curved hemostat are justified.

Thomas,[23] Leonardo,[12] Mosher,[3] McCaskey,[15] and Barnhill[1] have strongly emphasized the necessity for fearless surgery and Barnhill[1] says that deep neck infection is no place for the procrastinating surgeon. The cosmetic result is a minor issue and should never prevent adequate measures being taken to provide the best drainage possible. For submaxillary abscess the incision should begin just anterior to the angle of the jaw and pass forward parallel to and about 1 cm. below the mandible for three-fourths of the distance to the midline anteriorly. It will divide the skin, platysma, cervical fascia, mylohyoid muscle and the anterior belly of the digastric muscle. The only large vessels encountered are the facial artery and vein as they pass up the posterior lateral side of the submaxillary gland; these are isolated, ligated, and cut. The gloved finger is inserted and passed upward, forward and backward in search of an abscessed pocket. Two soft rubber drains are sutured into the depth of the incision and partial closure made.

When massive swelling is present, with involvement of the sublingual space as well as the submaxillary space, the original incision extends to the midline. When it is suspected that the infection lies above the geniohyoid, the muscle is incised. Now a curved Kelly hemostat is inserted through the incision, anterior to the submaxillary gland, and passed upward toward the floor of the mouth. When the mouth can be opened, it is well to place the gloved finger in the floor of the mouth and to palpate the end of the hemostat as it passes upward by blunt dissection, avoiding perforation of the oral mucosa. A fold of rubber tissue is inserted to the bottom of the cavity and partial closure made around the primary wound. If the infection is bilateral, the incision should extend continuously around to near the

angle of the jaw on the opposite side, dividing all the fibers of the mylohyoid muscle. Whether one or both sides are involved, marked relief from tension follows division of the mylohyoid diaphragm, allowing the tongue and the edematous tissue above to descend and thus the natural airway is regained. Mosher[3] advocates a T-shaped incision over the submaxillary area as his approach to deep infections in the neck and this incision is excellent for the exposure and drainage of the involved spaces.

Furstenberg[21] says that the treatment of suppurative disease of the parotid calls for massive drainage of the gland. He obtains this best by an incision which starts above near the zygoma, immediately in front of the ear, extending downward to a point beneath and well in front of the angle of the jaw. The skin and subcutaneous tissue are then reflected forward, permitting a wide exposure of the entire gland. Incision may be made to a considerable depth within the gland without danger of injury to the facial nerve, as these fibers lie rather deep. Since the parotid gland is composed of compartments or acini, a stab incision does not afford proper drainage of a suppurative gland. Folded rubber drains are kept in the depth of the wound as healing progresses to the surface of the tissues.

For abscess in the pterygomaxillary space, an incision is made along the superior border of the zygoma and the fibers of the temporal muscle dissected to expose the infratemporal fossa. A grooved director or small hemostat is directed downward and inward under the zygomatic arch for a distance of one to one and a half inches and entry made into the pterygomaxillary space. A small rubber tissue drain is inserted and partial closure made. When extension has taken place into the mandibular fossa, the articular capsule may be loosened. Care must be taken not to enter the joint proper, as ankylosis may result.

When the lateral pharyngeal space is involved secondarily after operative treatment on the submaxillary and sublingual areas, the original incision is carried directly backward and slightly upward, following the curve of the mandible, to the anterior border of the sternomastoid muscle. Many surgeons prefer the T-shaped incision of Mosher,[3] particularly when the principal area of involvement is in the lateral pharyngeal space. After division of the superficial tissues and cervical fascia, Barnhill[1] selects his entrance on a line from the middle of the submaxillary gland to the middle of the parotid.

A dull dissector is inserted and passed inward and upward in the direction of the tonsillar fossa, and an index finger in the pharynx, placed just anterior to the tonsil, will detect the end of the approaching instrument. The finger may be used instead of the blunt dissector and when the bottom of the space is reached we have internally only the constrictor muscle of the pharynx, covered externally by the buccopharyngeal fascia and internally by mucosa. The wound should be left fairly well open and two or more folds of rubber tissue give excellent drainage, when placed in the bottom of the abscessed cavity.

When the posterior compartment of this space has become involved and it is necessary to investigate the carotid sheath, or its contents, an incision is made along the anterior border of the sternomastoid muscle from the mastoid tip downward as far toward the clavicle as necessary to expose healthy tissue. The superficial layer of the deep cervical fascia is exposed and cut through along the anterior edge of the muscle, far enough forward so as to include the two layers after they pass around the muscle and unite again. Blunt dissection is made between the carotid sheath and the deep layer of fascia, which passes under the muscle, the connecting loose areolar tissue being easily divided with the finger. Abscess just outside the carotid sheath is evacuated as soon as the sheath is reached. Adjacent infected lymph nodes in the operative field may be removed. When the carotid sheath presents even slight pathological changes, it should be opened and its contents inspected. Particular attention is given to the internal jugular following previous intermittent chills and a septic type temperature. If the vein is thrombosed, the limits of the clot are determined and the vessel is ligated above and below the involved area. If the thrombus in the internal jugular is below the entrance of the facial vein, this should be ligated and the diseased portion of the internal jugular excised and removed. A large cigarette drain is placed in the wound, and it is closed except for the inferior extremity.

Orton[24] states that in approaching the buccopharyngeal fascia, after exposure of the carotid sheath, the vessels are retracted outward and the trachea and the thyroid gland inward. The space beneath the fascia is entered by blunt dissection and since infection may extend downward to the mediastinum, complete exploration for an abscess is made. When good exposure has been secured, a rubber dam drain is inserted and the wound left fairly well open until it is determined if spreading of infection is taking place. When it becomes necessary to do a cervical mediastinotomy, after the procedure of Furstenberg,[21] it is well to remember that the esophagus is in close

relationship with the pleura high up to the left side, while on the right the esophagus and pleura come in contact at a lower level leaving more room above for exploration and insertion of drains either anteriorly or posteriorly.

When there is marked difficulty in respiration at any stage of these deep infections, early tracheotomy should be considered. This may be performed before surgical drainage, at the time of operation, or postoperatively. The majority of patients with any serious impairment of respiration would be much safer, and the prognosis more favorable, if a tracheotomy were performed before surgical drainage of the infected area. The most dependable way to secure an adequate airway is to perform a tracheotomy. Hart[25] states that this should be done before the administration of any inhalation anesthetic because the irritation of the anesthesia may lead to spasm of the laryngeal structures with fatal occlusion. He states further that intubation is not recommended, because in itself it may lead to spasm before surgical drainage can be completed. When the respiratory rate increases and a decrease in the depth of respiration is noted, associated with change in the voice and occasionally with cyanosis, a tracheotomy should be done. A tracheotomy does not increase the danger of spread of the infection and has negligible bearing on the occurrence of pneumonia. It should always be performed under local anesthesia. Oxygen should be available for immediate use at any time deemed necessary. In the fulminating infections, especially where convalescence is prolonged, repeated blood transfusions are a great help.

The type of anesthesia to be used is of very great importance. Local anesthesia is satisfactory in uncomplicated cases of mild infection and is adequate for simple incision and drainage. It can be used, if necessary, in the deeper infections when a general anesthesia is contraindicated, but there is the risk of spreading the infection and also shock to the patient is more marked. Some men use infiltration and block anesthesia for practically all cases and report good results. Inhalation anesthesia should always be used with great caution in severe infections and under certain conditions should never be used. When encroachment on the airway is limited to the tongue and the pharyngeal region and when this can be overcome by the insertion of a pharyngeal breathing tube, a general anesthetic is fairly safe. Laryngeal spasm may occur here and necessitate interruption of the operation and immediate tracheotomy. Helium gas, mixed with the anesthetic agent, gives a more even anesthesia and causes less irritation. When an expert anesthetist cannot be obtained and a general anesthetic is deemed advisable, the trachea should be exposed before

beginning surgical drainage. When actual dyspnea is present before the operation, general anesthesia is absolutely contraindicated until the airway has been restored. Inhalation anesthesia can be given successfully following tracheotomy if there is no involvement of the lungs. When the condition of the patient is such as to warrant the use of a general anesthetic, cyclopropane or ethylene gives good results. Surgeons have warned repeatedly against the use of nitrous oxide and ether because of the high instance of spastic respiratory obstruction.

Intravenous anesthesia has been used with much success and offers great advantages. Spastic blockage of the airway rarely occurs, the patient recovers consciousness rapidly and without the ill effects of a general anesthetic, and the surgeon is free to work without interruption by the anesthetist. Both evipal and sodium pentothal have been widely used and few fatalities reported. Experience is teaching that these intravenous barbiturates are probably the safest anesthetic to use in deep infections of the neck. Operative procedures on these conditions are always performed with great risks under any type of anesthesia. A complete tracheotomy set and a Mosher lifesaver laryngeal cannula should always be kept at hand.

CONCLUSIONS

1. Early request of consultation and aid by the dental surgeon will do much to avoid the serious consequences of a deep infection of dental origin.

2. Prophylactic care of the teeth and gums in all cases of advanced infection should precede surgical procedure. Sulfonamide preparations or penicillin for several days in advance of surgery on severe oral and dental infections would help in preventing complications.

3. The molar teeth are most frequently at fault when the neck is secondarily involved, and mandibular injection is frequently the source of infection.

4. Intravenous barbiturates have proven highly satisfactory in producing adequate and safe anesthesia.

5. When cases are seen late and the deeper structures of the neck are involved, with dyspnea present, the paramount issue is to establish immediately an adequate airway; next, to drain the infected area.

706 FLATIRON BUILDING.

REFERENCES

1. Barnhill, J. F.: Deep Abscess of the Neck, Surgical Treatment, Am. J. Surg. 42:207-219 (Oct.) 1938.

2. Blassingame, C. D.: Angina Ludovici: An Anatomical and Clinical Study, Arch. Otolaryng. 8:159-176 (Aug.) 1928.

3. Mosher, H. P.: The Submaxillary Fossa Approach to Deep Pus in the Neck, Tr. Am. Acad. Ophth. and Otolaryng. 34:19-36, 1929.

4. Grodinsky, Manuel: Ludwig's Angina, Surgery 5:678-697 (May) 1939.

5. Iglauer, Samuel: Surgical Approaches to Deep Suppuration in the Neck and Posterior Mediastinum, Arch. Otolaryng. 21:707-716 (June) 1935.

6. Shure, I. I.: Abscess of the Pterygomaxillary Space with Involvement of the Mandibular Fossa, Arch. Otolaryng. 38:230-235 (Sept.) 1943.

7. Greenfield, S. D.: Pterygomaxillary Abscess Associated with Acute Suppurative Otitis Media, Simulating Zygomatic Mastoiditis, Laryngoscope 44:232-239 (March) 1934.

8. Orton, H. B.: Deep Infection of the Neck, J. A. M. A. 120:873-879 (Nov.) 1943.

9. Trout, H. H.: Ludwig's Angina, Arch. Surg. 41:532-571 (Aug.) 1940.

10. Taffel, Max, and Harvey, S. C.: Ludwig's Angina, Surgery 11:841-850 (June) 1942.

11. Muckleston, H. S.: Angina Ludovici and Kindred Affections, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 37:711-735 (June) 1928.

12. Leonardo, R. A.: Ludwig's Angina: Review of 21 Cases, J. Intern. Coll. of Surg. 3:82-86 (Feb.) 1940.

13. Williams, A. C.: Ludwig's Angina, Surg., Gyn. and Obs., 70:140-149 (Feb.) 1940.

14. Tschiassny, Kurt: Ludwig's Angina: An Anatomical Study of the Role of the Lower Molar Teeth in its Pathogenesis, Arch. Otolaryng. 38:485-496 (Nov.) 1943.

15. McCaskey, C. H.: Ludwig's Angina, Arch. Otolaryng. 36:467-472 (Oct.) 1942.

16. Beck, A. L.: Deep Neck Infections, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 48:940-998 (Dec.) 1939.

17. Beck, A. L.: A Study of Twenty-Four Cases of Neck Infection, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 42:741-777 (Sept.) 1933.

18. Alden, A. M.: Deep Neck Infections Secondary to Dental Disease, J. Am. Dental Assoc. 23:854-860 (May) 1936.

19. Van Wagenen, W. P., and Costello, C. V.: Sublingual Phlegmon, Ann. Surg. 87:684-703 (May) 1928.

20. Barnhill, J. F.: Surgical Anatomy of the Head and Neck, Baltimore, William Wood and Co., 1937.

21. Furstenberg, A. C.: Acute Suppurations of the Mouth, Pharynx, and Neck, New Orleans Med. and Surg. J. 94:162-169 (Oct.) 1941.

22. Gaines, T. R., and Hatcher, M. B.: Penicillin in the Treatment of Patients with Deep Infections of the Neck, Arch. Otolaryng. 42:1-5 (July) 1945.

23. Thomas, T. T.: Ludwig's Angina, Ann. Surg. 47:335-373 (March) 1908.

24. Orton, H. B.: Deep Infections of the Neck, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 47:48-54 (March) 1938.

25. Hart, V. K., and Jones, Ira: Ludwig's Angina, Bull. Charlotte Memorial Hosp. 1:24-27 (June) 1944.

IV

SUPPURATIVE LABYRINTHITIS

NELSON A. YOUNGS, M.D.

AND

J. R. LINDSAY, M.D.

GRAND FORKS, N. D.

Labyrinthitis of tympanic origin has been classified into four main groups: (1) circumscribed, (2) diffuse serous, (3) diffuse manifest suppurative and (4) latent suppurative.

The circumscribed type, sometimes referred to as perilabyrinthitis, is indicated by recurrent vertigo in the presence of varying degrees of function and a positive fistula reaction. It occurs mainly in cases of cholesteatoma or granulomatous inflammation and does not enter the discussion in this paper.

Serous labyrinthitis is differentiated from the suppurative types by the presence of demonstrable function. The terms "paralabyrinthitis" and "labyrinthine irritation" have sometimes been used in connection with mild labyrinthine symptoms without impairment of functon, but may for practical purposes be included under the heading "serous."

"Latent suppurative" refers to the quiescent stage following the acute or "manifest" stage of diffuse suppurative labyrinthitis. Total loss of function is the criterion for diagnosis of a suppurative type.

The grave significance of invasion of the inner ear by infection is indicated by the high mortality in former years. Previous to the development of petrous pyramid surgery and sulfonamides cases of fatal meningitis were reported in as high a proportion as 30 per cent in some clinics.

The clinical differentiation of suppurative and serous labyrinthitis is not always easy to determine. In severe stages of the serous type function may be depressed to the point where it cannot be demonstrated with certainty, thereby suggesting the suppurative

---

From The Grand Forks Clinic, Grand Forks, North Dakota, and the Department of Surgery (Division of Otolaryngology) of The University of Chicago.

Read at the Middle Section Meeting of the American Laryngological, Rhinological and Otological Society, Minneapolis, Minn., Jan. 16, 1946.

stage. Only after the acute symptoms have passed may the return of a small degree of function indicate that the lesion was of the serous type. Histologically it is common in such cases to find inflammatory changes with connective tissue proliferation in the perilabyrinthine spaces with varying degrees of degeneration of the neural elements. The endolymphatic spaces may show dilatation (hydrops) but the membranous labyrinth appears to have remained intact and the endolymphatic spaces free from inflammatory changes.

A similar histologic picture may also be found when function has apparently been totally destroyed. The inflammatory reaction may result in an advanced degeneration of the neural elements with total loss of function without actual invasion of the labyrinth by the infecting agent. Surgery in such a labyrinth would be associated with the same danger as opening a normal labyrinth in the presence of middle ear suppuration.

Because no reliable clinical indication exists as to the presence of suppuration within the labyrinth Friesner and Rosenwasser[1] preferred to class "serous" and "suppurative" labyrinthitis under one heading "acute labyrinthitis." Lund[2] preferred the terms "acute destructive" and "chronic destructive" instead of "manifest" and "latent suppurative" since the loss of function might be caused by a degeneration of neural elements.

A diffuse suppurative labyrinthitis is known by histopathologic observation to produce rapid invasion of the membranous labyrinth and destruction of the sensory epithelium.

The clinical differentiation of a suppurative from a serous type on the basis of total loss of function is therefore not a reliable criterion of the pathologic picture and hence of the necessity of labyrinthine surgery. The presence of function has, however, been a reliable indication that the labyrinth need not be opened.

Opinions regarding the management of labyrinthitis with total loss of function have varied. The Vienna School represented the more radical view and advocated a labyrinth operation in all cases of the manifest suppurative type. Since loss of function does not always mean frank suppuration, a number of labyrinths would by this policy be opened unnecessarily. The conservative view represented by such reports as that of Lund[2] and Lillie[3] has been more widely followed in recent years. In general, this policy has been to reserve the labyrinth operation until signs of meningeal irritation or spinal fluid changes could be detected.

During recent years the treatment and prognosis of labyrinthitis has been radically influenced by two major developments in the management of otitic suppuration.

The first consisted in the demonstration that the development of a labyrinthitis from acute or simple chronic middle ear suppuration was frequently due to a focus of suppuration in the petrous pyramid which had inadequate drainage.[4, 5] Invasion of the labyrinth in degrees varying from simple irritation to destruction and sequestration has been observed in approximately 25 per cent of such cases by one of us.

The surgical drainage of the focus of suppuration outside the labyrinth is the primary consideration in the prevention of an intracranial extension in these cases, and may if promptly carried out obviate the destruction of function.

Conservatism in the management of labyrinthitis has been further reinforced by the development of sulfonamide and penicillin therapy. Experience with these drugs suggests that a labyrinth operation will rarely be indicated except in the presence of a suppurating fistula or sequestration. The ability of these drugs to cure a pneumoccocus or streptococcus meningitis after a labyrinthitis has developed appears to support this view.

The relation of an undrained focus of suppuration in the pyramid to invasion of the labyrinth, and the relative place of surgery and chemotherapy in the management of such cases is illustrated by the following case report.

REPORT OF A CASE

D. K., white, female, aged 14, was seen April 30, 1942. She had had a discharging right ear for two weeks, dizziness, nausea and vomiting for four days, and pain in the right side of the head, centering around the right eye, for five days.

Physical examination revealed an ataxic patient with a spontaneous nystagmus toward the left. The right external auditory canal was filled with pus coming from an attic perforation. Mastoid tenderness was marked. The roentgenograms revealed bone destruction throughout the right mastoid. The petrous pyramids were pneumatized. The cells in the right apex were clouded, but there was no apparent bone destruction.

On May 1, 1942, a right simple complete mastoidectomy was done. The patient did well, her nystagmus cleared up and her pain

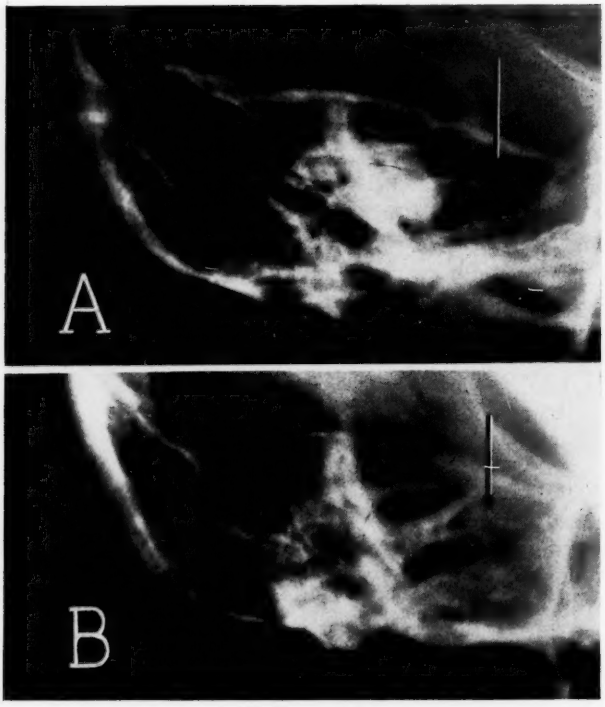


Fig. 1.—Roentgenograms of the petrous pyramids by the Stenvers projection two weeks after the onset of disease. *A*. The left or normal side shows pneumatization of the upper part of the pyramid,while *B*. the diseased side shows clouding of the corresponding area.

disappeared until the evening of the third postoperative day when she developed headache, partial paralysis of the right side of her face and a nystagmus. These symptoms all became progressively worse. On the fifth postoperative day the spinal fluid was under 380 millimeters of water pressure with normal response to jugular compression on each side. The fluid contained 377 cells of which 76 per cent were polymorphonuclears. Spinal fluid cultures were sterile.

Two days later she developed sharp precordial pain accompanied by spiking temperature. At this time a definite precordial rub could be heard over the pulmonic area. These findings indicated that the patient might be developing a blood stream infection by way of the

lateral sinus. The internal jugular vein was therefore ligated. At the same time the lateral sinus was investigated and found not to contain an obliterating thrombus. However, daily improvement was noted after this procedure.

On the eleventh postoperative day there was some return of motion to the right side of her face. On the nineteenth hospital day the right drum head and the postauricular incision had healed. The patient was discharged from the hospital on her twenty-sixth hospital day.

TABLE 1.—SUMMARY OF LABORATORY FINDINGS.

DATE	SPINAL FLUID						BLOOD			
1942	P.	CELLS	PMN.	LYM.	SUGAR	CULT.	HGB.	WBC	S.R.	S.L.
4/20							80	12450	74	
5/2							75	12400		
5/4							72	12200		8.9
5/5							76	11700		11.8
5/7	380	377	76	24	88	Neg.	52	17200		20.0
5/8							74	17500		12.8
5/10							66	14400		11.2
5/16	220	187	38	62	73.4	Neg.	75	6600		10.3
5/25	190	152	26	74	74	Neg.				

In the above table P. stands for spinal fluid pressure in mm. of water; S.R., for sedimentation rate millimeters in 60 minutes; S.L., for the sulfadiazine level measured in milligrams per 100 cc. of blood.

She had been on sulfonamide therapy for a number of days before being referred to me. Throughout her hospital stay the blood sulfadiazine levels were maintained between 10 to 20 milligrams per 100 cc. The organism recovered from the mastoid was hemolytic streptococcus. After each surgical procedure the mastoid cavity was filled with sulfanilamide and a small rubber drain was left just beneath the skin.

A month after the patient was discharged from the hospital the labyrinthine function was tested by means of the cold caloric test.

The left ear responded after douching with cold water for 45 seconds. There was no response after douching the right ear for over three minutes with the water at 9° C.

At that time her audiogram (Fig. 2) showed some hearing in the right ear using maximum masking obtainable with the Maico audiometer. The Weber test was lateralized to the involved ear, and

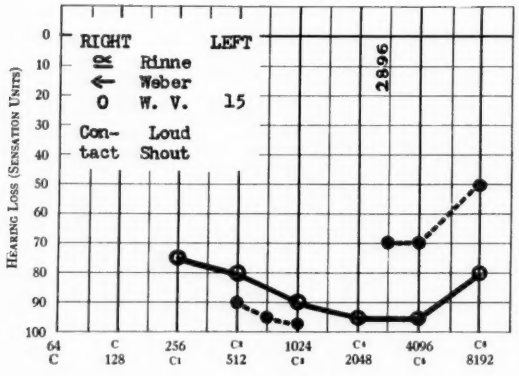


Fig. 2. Audiogram—right ear. Solid lines represent hearing curve obtained April 30, 1942. Broken lines represent hearing curve obtained July 1, 1942. Maximum masking obtainable on the Maico audiometer was used to mask out the good ear. Tuning fork and voice tests are of interest in that repeatedly the patient heard the 256 fork by bone conduction over the right mastoid process for a longer period of time than on the normal side. (A Bárány noise apparatus was used in the voice test.)

the 256 tuning fork was heard by bone conduction over the right mastoid process for a longer period of time than over the left.

Throughout the next year the patient was seen in the office periodically. She remained well, attended school, passing all her subjects with grades of A and B.

On June 6, 1943 (a year and two weeks after her previous discharge from the hospital) she was readmitted because of severe pain in the right ear, radiating to the supraorbital region, and a paralysis of the right side of her face.

Physical and neurological examinations were negative except for a right-sided facial paralysis of the peripheral type. The right drum head was intact and free from inflammation.

The impression was that she had a Bell's palsy and an associated vidian neuralgia of unknown etiology. She was treated with large doses of thiamine chloride and local application of heat. In a few days the pain subsided and the patient was discharged from the hospital. Her facial paralysis persisted.

The patient again entered the hospital August 21, 1943 (a year and three months after her first admission) with marked nausea, vomiting, vertigo, diplopia and a painful discharging right ear of six days' duration.

Examination revealed a pulsating discharge in the right external auditory canal, a right-sided facial paralysis of the peripheral type, nystagmus, which would change with the direction of gaze but seemed most prominent upon looking toward the left, and diplopia in all meridians. Neurological and physical examinations were otherwise negative. Spinal puncture revealed the fluid to be under 225 millimeters of water pressure. There were 68 cells of which 65 per cent were lymphocytes. The sugar was 145 milligrams per 100 cc. Spinal fluid cultures were sterile.

The patient did not improve under conservative treatment. On August 25, 1943, the mastoid was opened, and the region of the labyrinth searched for a fistula or cell tracts. Some perilabyrinthine bone necrosis was found but no other lesion.

On Augut 28 about one diopter of edema of each nerve head was noted. Asynergy as shown in the finger-to-nose test with the right hand was also present. Spinal puncture revealed the fluid to be under 535 millimeters of water pressure. There were 257 cells present, 75 per cent of which were polymorphonuclears; the sugar content was too low to compare with a standard, and the cultures revealed the presence of a hemolytic streptococcus.

The patient failed to respond to chemotherapy, blood transfusions, and surgery. She expired September 18, 1943, three weeks after the onset of her meningitis.

TABLE 2.—SUMMARY OF LABORATORY FINDINGS.

DATE	SPINAL FLUID						BLOOD		
1943	P.	CELLS	PMN.	LYM.	SUGAR	CULT.	HGB.	WBC	S.L.
8/21	210	68	35	65	142	Neg.	90	13100	
8/26							79	26000	8
8/29	525	257	72	28	0	H.S.	72		
9/1	550	3990	95	5	0	H.S.		33600	10
9/5	680	909	83	17	0	H.S.	64		13.3
9/10	550	1369	94	6	0	H.S.	48		20
9/14	450	708	88	12	0	H.S.	60	50600	10.9
9/16	200	3765	95	5	0	H.S.			11

In this table P. stands for spinal fluid pressure measured in mm. of water; S.L. for the sulfadiazine level in the blood as measured in milligrams per 100 cc. of blood; H.S. for hemolytic streptococcus.

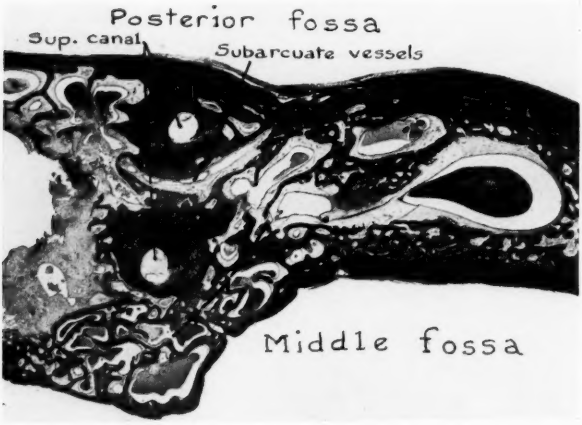


Fig. 3.—Horizontal section through the right pyramid passing through the superior semi-circular canal at the level of the subarcuate vessels and immediately above the geniculate ganglion. The arrow indicates the route by which the large air cells in the pyramid had developed from the epitympanum and which provided the only natural outlet. The cells have a thickened lining mucosa and are filled with exudate. The bone shows an advanced degree of sclerosis, the marrow spaces being obliterated in part and filled with fibrous tissue.

Fig. 4.—Horizontal section at a lower level than Fig. 3. This passes through the basal coil of the cochlea, the ear drum, the horizontal portion of the carotid artery and the lower edge of the internal auditory meatus. The chronic sclerosing osteomyelitis seen in the upper levels has given way to extensive necrosis and suppuration bordering on the carotid artery. The large bony sequestra (S) enclosed in a dense fibrotic wall lie medial to and encroach on the internal meatus.

The postmortem examination revealed a diffuse meningitis more pronounced in the basilar areas and a walnut-sized brain abscess located in the extreme anterior tip of the right temporal lobe. The right temporal bone was removed for histopathologic examination.

Histopathologic Examination. The right temporal bone was removed, prepared, and sectioned serially in the horizontal plane.

Middle Ear and Mastoid. The middle ear and the eustachian tube mouth showed moderate inflammatory reaction of the mucosa and were filled with exudate.

The mastoid cells had been all removed with the exception of a few cells external to the arcuate eminence. There were no air cell tracts leading from the mastoid into the pyramid.

The epitympanic space contained a number of cells filled with exudate and swollen mucosa from which a tract of cells extended between the superior canal and the geniculate ganglion into the pyramid.

Petrous Pyramid. The tract of cells leading from the epitympanum expanded in the region medial to the arcuate eminence into a group of large cells which occupied the posterosuperior angle of the pyramid well into the apex. (Fig. 3) These cells were filled with exudate; their mucoperiosteal lining was thickened, and the bony walls surrounding them extensively sclerosed.

The marrow spaces adjacent to the air cells showed changes of a chronic nature, mainly fibrosis and bony sclerosis. At a slightly lower level these chronic changes became interpersed with areas of bone necrosis and acute suppurative changes. In some areas spicules of new bone had been deposited.

The portion of the apex bordering on the internal auditory meatus was the site of two large bone sequestra surrounded by thick fibrotic walls. This chronic abscess cavity occupied a large part of the internal meatus. The tip of the apex was absent from the specimen and the site of meningeal extension therefore not definite. The bony wall of the horizontal part of the carotid canal was completely eroded so that the artery bordered on the suppurative focus. This would suggest that the extension to the meninges had followed the course of the artery to the middle fossa. (Fig. 4)

Inner Ear. The cochlea was involved by an inflammatory process which was partly chronic and partly acute.

Fig. 5.—Section through the upper half of the basal coil of the cochlea and the vestibule. The cochlea at this level shows early inflammatory changes. The 8th nerve is almost completely degenerated as is the spiral ganglion. Cellular infiltration of the scala tympani is beginning by way of the modiolus and Rosenthal's canal. The cochlear duct shows disintegration in the whorl at the left but is intact at the right. Its absence in the middle coil is artefact. Corti's organ is absent. The vestibule shows only mild inflammatory changes. The defect in the membranes is probably artefact.

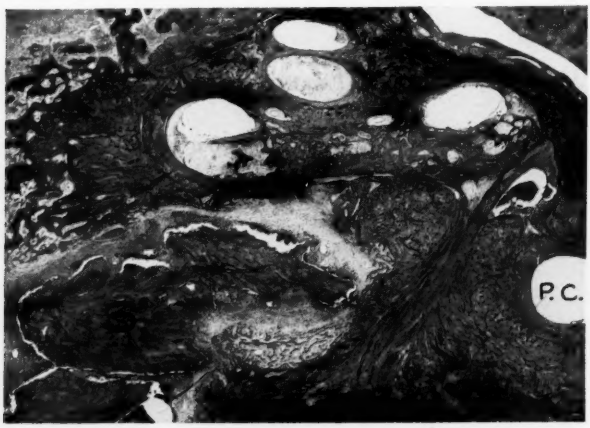


Fig. 6.—Higher magnification of Fig. 4. The sequestra (S) include part of the cochlear capsule bordering on the scala tympani which has been almost obliterated by ossification. This represents the point of the initial invasion of the labyrinth which had been well localized. The ampulla of the posterior canal (P.C.) shows no inflammatory changes.

The otic capsule bordering on the basal coil had been eroded, the bone forming part of a sequestrum. The scala tympani of the lower turn of the basal coil was the site of a diffuse labyrinthitis ossificans, with some fibrosis extending into the upper turn.

The membranous labyrinth had apparently been intact until shortly before death. An acute inflammatory reaction with early cellular exudation was evident in the scala tympani of the upper turn of the basal coil with beginning disintegration of the walls of the cochlear duct, but in some areas the duct was still intact and of normal contour. A small amount of amorphous exudate was present in the perilymphatic spaces of the vestibule and semicircular canals. The membranous labyrinth was intact in the canals but the walls of the saccule and the utricle were in part absent apparently due to artefact. The sensory organs in the vestibule and ampulla were poorly preserved owing apparently to artefact and inflammatory changes. The spiral ganglion appeared to be completely degenerated as well as most of the cochlear nerve and Corti's organ. In the lower half of the basal coil Rosenthal's canal was obliterated by new bone and above that by loose connective tissue and cellular exudate. The vestibular nerve and Scarpa's ganglion also showed extensive degeneration. The abscess cavity encroaching on the internal meatus probably accounted for the loss of function of both the vestibular and facial nerves. The degeneration of the spiral ganglion in the basal coil dated from the first invasion of the labyrinth, but it is possible that some function had been present in the middle and upper coils until the final attack.

SUMMARY AND INTERPRETATION

The acute suppurative process in the middle ear spaces invaded the petrous apex by way of a pneumatized tract from the epitympanum.

Exenteration of the mastoid could not provide drainage from the apex in this case. The disease in the apex, consisting of suppuration in the air cells and osteomyelitis, resulted in formation of sequestra and a serous labyrinthitis which was localized mainly in the basal coil of the cochlea.

The effect of sulfadiazine therapy was sufficient to relieve all acute symptoms and produce an apparent cure. On the basis of the histological picture it appears that the clinical evidence of persistent remnants of function may have been correct.

The presence of osteomyelitis with sequestration was responsible for persistence of the infection which, although localized for about a year, eventually invaded the meninges.

The labyrinth had been the site of an acute inflammatory process on two occasions; the first shortly after the onset of the illness, and the second in the terminal stages. Clinically, the classification of the process as serous or suppurative presented difficulty but the pathologic analysis proved that the invasion of the labyrinth was coincidental and of secondary importance. The meningitis was a direct extension of the suppurating focus in the apex. Surgery of the labyrinth was not indicated and could only have increased the danger to the meninges.

This case is an example of the complicated clinical picture which may accompany a petrous pyramid suppuration and illustrates the limitations of chemotherapy in its present form. In retrospect it is obvious that removal of sequestra such as these would scarcely be feasible but surgical drainage of the apex combined with chemotherapy could in such cases be expected to obviate meningeal complications.

GRAND FORKS CLINIC.

REFERENCES

1. Friesner, I., and Rosenwasser, H.: Treatment of Labyrinthitis, Arch. Otolaryng. 20:139 (Aug.) 1934.

2. Lund, R.: Indications for the Labyrinth Operation with Special Reference to Acute Diffuse Descriptive Labyrinthitis, J. Laryng. & Otol. 51:425-452 (July) 1936.

3. Lillie, H. I.: Critical Review of Patients Subjected to Labyrinth Operations, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 53:207-220 (June) 1944.

4. Jones, Marvin: Disturbed Vestibular Function: Causes and Cures, Arch. Otolaryng. 41:272 (April) 1945.

5. Lindsay, J. R.: Textbook on the Nose, Throat and Ear, and Related Diseases, Edited by Chevalier Jackson, M.D. and Chevalier L. Jackson, M.D., Philadelphia and London, W. B. Saunders Company, 1945.

V

SECRETORY OTITIS MEDIA

WATT W. EAGLE, M.D.

DURHAM, N. C.

The disease which we now commonly term "secretory otitis media" is in reality no new disease. Many years ago the favorite term for the disease was "catarrhal otitis media"; later "nonsuppurative otitis media," "hydrops of the middle ear," and "tubotympanic catarrh," as well as many other synonyms evolved. Whichever term is chosen, it is self-explanatory of the disease process.

Catarrhal otitis media was recognized clinically long before the electric otoscope was invented—in those days when sunlight rays were converged by a concave mirror into a speculum for inspection of the ear drum. Up to 1940 the disease had never been considered very common. However, in the past four years the disease has taken on a new significance, and this significance is due to the frequency with which it prevails. One could state that there has been a mild epidemic of "secretory otitis media," the term which we shall use for the disease henceforth in this paper.

The pioneer textbooks in the field of otolaryngology describe in detail the symptoms associated with secretory otitis media, and these symptoms are exactly the same as we find now. Thus, the purpose of this paper is not to bring forth a description of a new disease but to record our observations of an old disease, which for various reasons yet unknown has become quite prevalent in the past four years. The prevalence of the disease is not restricted to our immediate locality in the South, or even the entire United States. The Armed Forces scattered all over the United States were subject to this disease; in addition, other members of the Armed Forces in the European and Pacific Areas were also found to have the same condition, sometimes long after they had left this country.

Secretory otitis media is produced by an obstruction occurring in the eustachian tube, preventing drainage of the normal secretions

From the Division of Otolaryngology, Department of Surgery, Duke University Medical School and Hospital.

Presented before the Southern Section of the American Laryngological, Rhinological and Otological Society, Richmond, Va., Jan. 7, 1946.

of the middle ear and impairing ventilation of the middle ear. Apparently, there are no known influencing factors which produce the eustachian tube obstruction. Regardless of the condition causing the disease, we know that when the eustachian tube becomes blocked a vacuum occurs in the middle ear, producing a transudate of fluid. This collection of transudatory fluid in the middle ear, together with the normal mucoid secretions of the middle ear, is what we consider secretory otitis media. This fluid is almost invariably sterile, and it is rather viscid. The disease occurs in the white and negro races. However, since there is very little pain associated with the disease, it is probable that the reason fewer negro patients are seen is due to the fact that negro patients rarely see their physician until they are in "misery." The disease is found in all generations and decades of life. Changes in the climate, whether humid or dry, and changes in the altitude, whether sea-level or high, and even plane flying, allergic states, sulfonamides, and upper respiratory infections have not been definitely identified with the disease, although the older textbooks state that changes in altitude and climate probably cause it. It is true that we have seen more patients having the disease during the real winter months, but even so, many of them had had no previous upper respiratory infection. The older textbooks also stated that infections in the nose, pharynx and nasopharynx were the most likely causes of the disease. Since the fluid has been found sterile, it is quite probable that the eustachian tube obstruction has prevented the passage of bacteria through the tube to the middle ear in those individuals having an associated upper respiratory infection. We do know of certain individuals who, during their visits to the office, continually sniff the nose, that is suck air into the nasopharynx through the nose, and quite forcibly so, and some of these individuals are among our patients having secretory otitis media. In each case of the "sniffers" the fluid in the middle ear was full of bubbles. Also we have observed neoplasms of the nasopharynx possibly producing the disease.

The symptoms of secretory otitis media are practically the same in all cases. They are characteristic of no other disease. They include the following: slight, if any, pain at any time; a feeling of fullness in the ear; sensations of bubbles or fluid moving in the ear, especially if the head is moved forward or sideways or when the nose is blown; tinnitus of a peculiar type, which is quite different from tinnitus due to other causes and is usually described as sounding like the telephone wires singing on a midwinter day; autophony, or the sensation of hearing oneself talk; numbness and a sensation of deadness over the entire side of the head, especially noted in the region just above and behind the ear; grating sensations as the hair is combed; dizziness,

Fig. 1.

A—Fluid level.
B—Fluid level, with peak at umbo.
C—Fluid level with air bubbles in fluid.
Frequently the entire middle ear is filled with fliud—including bubbles.

which usually is mild and usually not associated with nausea or vomiting; and most frequent of all, and most annoying to the patient, impaired hearing of mild degree (however, sometimes the hearing is more impaired for clarity than acuity). One pathognomonic sign is the clearing of the impaired hearing when the head is turned forward or sideways, only to have the impairment recur when a normal head position is resumed. It is probable that the fluid overlying the round window causes the impairment. In a majority of these patients there is some redness and rawness of the inner third of the auditory canal, and the canal bleeds very freely if barely touched by the instruments during examination or treatment. Occasionally there appears to be some granulation tissue in the auditory canal, but the entire inflammatory condition in the auditory canal clears up after the fluid has been aspirated from the middle ear.

The diagnosis of secretory otitis media is frequently made by the history the patient gives. It usually follows the pattern of the symptoms just enumerated. The inspection of the ear drum usually reveals what could be considered a normal, or fairly normal, ear drum, except in those cases of considerable duration where there might be slight retraction of the ear drum (Fig. 1). The light cone is sometimes diminished in brilliance but usually it is wider and dispersed. A fluid level in some cases is found, and this fluid maintains its level as the head is shifted forward or backward, the fluid acting similarly to the alcohol bead in the carpenter's level. In some patients the fluid level is straight across, while in others there is an elevation of the

fluid level up to a peak at the umbo. The fluid may reveal yellowish whorls similar to those seen in an agate marble, and this is especially true where there is no fluid level. On inspection of the ear, occasionally one is not sure there is any fluid in the ear, because the entire middle ear space is completely filled with the fluid. There is no bulging of the drum. Usually, however, in such cases there is a bluish-gray and dull appearance of the space behind the drum. In such cases with a typical history one is justified in incising the drum and usually the fluid is found. There are some cases where bubbles of air are found throughout the fluid. These patients are usually those individuals who violently sniff the nose or those patients recently having had inflations of the eustachian tubes. One can always be certain on inspection of the ear drum that he is not dealing with a purulent infection.

Other examinations, such as hearing tests and eustachian tube inflations, are aids in reaching a diagnosis. Roentgenograms are of no value whatever, since there is no destruction or even inflammation in the mastoid cells. It is possible for general practitioners to make the diagnosis from the history, but we should not hold them responsible for making a diagnosis when the history is questionable or the diagnosis depends on the appearance of the drum and the middle ear cavity. The practitioner is not accustomed to differentiating such a disease as secretory otitis media and he may be perplexed when he finds a normal drum. Therefore, some patients have had the disease for several weeks or months, or even years, when they are referred or voluntarily go to an otologist.

The treatment in the past decades for secretory otitis media has been on the conservative side to a detrimental state. We speak from personal knowledge and experience, as will be shown in some of our case reports. In all of the texbooks of the past and in most of the present textbooks, one is advised to use heat, various types of drops locally in the ear, to inflate the middle ear by the Politzer or Valsalva methods, and then if necessary to inflate after catheterization. Vibration of the drum by means of a Siegel otoscope has been recommended. Rarely has myringotomy been recommended in the past.

Our present-day method of treating secretory otitis media lies mainly in the fact that we *do* incise the drum. There is comparatively little pain on incising a normal ear drum—perhaps little more pain than is experienced during hypodermic needle injection—and it is to be remembered that in secretory otitis media we are dealing with an essentially normal ear drum. Therefore, no anesthesia is neces-

sary for the myringotomy, even in children, unless one is dealing with a very refractory child who must be held still during the procedure of drum incision and aspiration. If the fluid has been present for some weeks, the drum may be slightly retracted. As the myringotomy is performed, small bubbles of air gurgle into the middle ear, and these bubbles may be seen by the examiner and are heard by the patient. The fluid in the middle ear is always so thick that it will not exude from the opening and it is invariably necessary to aspirate the fluid from the middle ear by means of a small aspirating tip. This procedure is usually performed through the electric otoscope. The fluid has the appearance of castor oil or bee's honey, and it is so viscid that it may string out several inches. During the aspiration quite a swishing noise is made. Occasionally it is necessary to make another incision anteriorly in the drum to thoroughly aspirate the entire collection of fluid, since the counter-opening allows air to be pulled through the middle ear, the fluid emerging first into the aspirator tip. Immediately the patient notes restoration of hearing, loss of the singing type of tinnitus, loss of the dead or dull sensation over the side of the head, and the sensation of the ear being occluded. The retracted drum resumes its normal position.

The incision made in the drum need not be large and usually it will heal in a few hours' time. There is no bleeding from the incision since the drum is not inflamed. There is no recurrence of the fluid in most cases, and this is especially true when the fluid has been present only a few days prior to the incision and aspiration. If the fluid does recur, the patient will again recognize the same symptoms as experienced previously and will certainly return to the otologist to experience once again the sensation of a normal ear. There are a few instances where for some reason there may be recurrence of the fluid on several occasions, and on rare occasions, as will be cited later, there are some cases in which fluid recurs for a period of six to eleven months. However, in our experience, at least 75 per cent of our patients never had recurrence of fluid in the middle ear, and of the remaining 25 per cent there are only three patients who have required more than three additional myringotomies on the same ear.

It is rarely necessary to catheterize and inflate the eustachian tube. Apparently this fact is due to a spontaneous opening of the eustachian tube and continued normal function after the drum has been incised and the fluid aspirated from the middle ear. We found those patients on whom catheterization of the eustachian tube after aspiration was deemed necessary to be the very patients who had the prolonged disease, requiring numerous myringotomies.

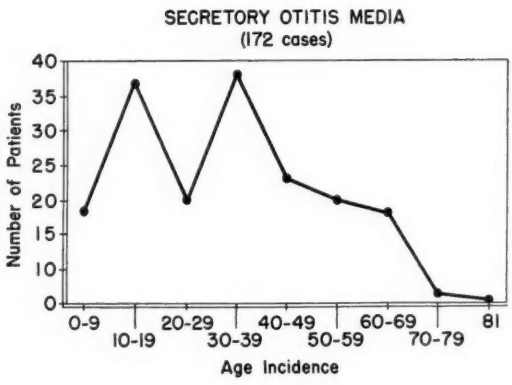


Fig. 2.

By using sterile technique for the myringotomies, one does not anticipate the onset of a purulent otitis media replacing the secretory otitis media. In only one instance did we see this occur. Since the disease is nonpurulent, one does not anticipate any mastoid involvement or other complication of any kind. In case the secretion is not removed by aspiration from the middle ear, there may eventually be an old adhesive otitis media with conductive deafness resulting but apparently this process usually takes many years. In such instances the drum becomes adherent to the other structures and walls of the middle ear.

The prognosis for secretory otitis media in the past has always been rather gloomy, and this was probably due to the conservative treatment used. In our experience we have observed no cases where the hearing was impaired permanently; as a matter of fact, even our most prolonged cases showed return of hearing to the status previous to the onset of the disease. We, therefore, do not consider the prognosis bad in any case of secretory otitis media. We do urge the use of 1% solution of ephedrine sulfate in physiologic sodium chloride or 0.25% neosynephrine hydrochloride as nose drops to keep the nasopharyngeal orifices of the eustachian tube shrunk in our effort to prevent a chronic tubal insufficiency. This medication is to be used immediately following aspiration of the fluid from the middle ear. We do not even find this treatment necessary in many cases. We also ask patients to refrain from sniffing the nose, which could cause negative pressure in the middle ear.

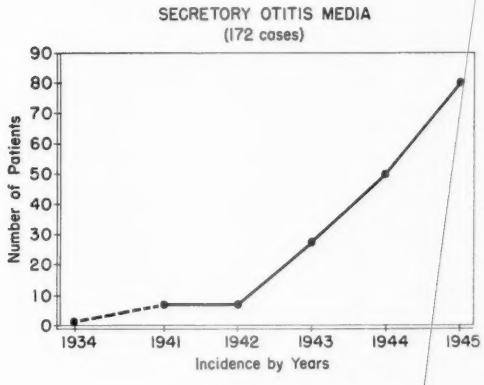


Fig. 3.

REPORT OF CASES

In this paper we wish to record our observations of secretory otitis media on 172 patients from the Private Service alone of Duke Hospital. All of these cases except one have been seen since 1940. These patients varied in age from 4 to 81. There were 99 males and 73 females. There were 70 cases of involvement of the right ear alone and 49 cases of the left ear alone, and there were 53 cases of bilateral secretory otitis media. There were a few patients listed with the bilateral group which showed only one ear affected originally, only some time later to have the other ear become involved. There were 3 cases where secretory otitis media existed in one ear and a purulent otitis media existed in the other ear.

SECRETORY OTITIS MEDIA
(172 cases)

		Bilateral	53
Males	99	Right ear	70
Females	73	Left ear	49
	172		172

The incidence relative to age groups showed that of the 172 total number of patients there were 18 below age 11, 37 patients were in their teens, 20 patients were in their twenties, 38 in their thirties, 23 in their forties, 20 in their fifties, 13 in their sixties, 2 in their seventies, and one patient was 81. (Fig. 2).

In 1934 one patient is recorded as having fluid in the middle ears. From that time until 1941 no patients were seen with this disease, and neither had there been any patients from 1930 (the time of the opening of our hospital) until the above-mentioned patient in 1934. During 1941 there were 7 patients seen with secretory otitis media; in 1942 also 7 patients were seen; but in 1943 the number of patients had risen to 27; in 1944 there were 50 patients; and in 1945 during the eleven months prior to December there were 80 patients seen (Fig. 3). It is difficult to believe that our eyes had failed us prior to 1941, or that we did not listen to patients' histories!

The average duration of symptoms of the patients seen was about equally divided between those having the disease less than one week and those having the disease from one week up to four years. The history in those cases of long duration is accepted, since we had personal experience, as cited in Case 2, where a patient had the disease over a period of four years. In our case reports to follow there are some of short duration and typical cases, while the others include our complete series of prolonged cases.

CASE 1.—M. M., male, age 37, October 1934. There was a typical history of secretory otitis media. Our notes state: "Two years ago he first noticed a sensation of noise in the ears while talking and could feel, as well as hear, himself talk." He was seen by two specialists previous to our examination and one of them had removed some spurs from the nasal septum to provide better breathing space. The patient developed a habit of sniffing forcibly through the nose, and when we saw him, the middle ears were filled with bubbles. He could inflate both ears quite well by the Valsalva method, which only served to churn the bubbles further. He would then have a sensation that the ears were full and would immediately sniff again, which seemed to him to open the eustachian tubes and deflate the middle ears. Our notes of 1934 further state: "He is to drink tumblers of water while holding the nose and if the fluid has not by its normal method left the middle ears within a few days, the drums should be incised and the fluid forced out of the middle ears by the Valsalva method of inflation." This patient did not return and we have no further notes regarding his case. This is a case of correct observation and diagnosis, but incorrect therapy—probably due to the old fear that had been instilled in us never to open what appeared to be a normal drum.

CASE 2.—J. R., male, age 39, February 1941. This patient was seen seven years after Case 1, no cases being seen in the intervening

years. Six months previously he had onset of symptoms without any preceding illness. Tinnitus had been present in both ears, combined with slight impairment of hearing. He had been subjected to twelve eustachian tube inflations before we saw him, all without benefit. During examination it was noted that he continually sniffed the nose and both middle ears were found to be filled with bubbles. Our notes of 1941 on this patient state: "Suspect it will be necessary to incise each drum and then inflate the tubes to get rid of the discharge by the inflations." He returned on several occasions and both eustachian tubes were catheterized and inflated, with the same resultant formation of bubbles. However, for some reason which we cannot now account for, we failed to follow our own advice and the ear drums were not incised. During the inflations there was a terrific rasping noise audible to the patient, as well as the examiner, on account of the formation of bubbles. Apparently he became tired and disgusted with receiving no benefit following the inflations; after three months he failed to return for any further inflations. In September 1945, over four years later, we wrote this patient to inquire as to the status of his ears and invited him to return if he was still having trouble with his ears. This invitation was extended because of the results we had obtained with numerous patients with this disease during the interim. He was quite willing to return and to our surprise both ears were found still to be filled with the same type of fluid and bubbles of secretory otitis media. The drums were incised and the fluid was aspirated. There was immediate relief of the symptoms of secretory otitis media for the first time in more than four years, and he was indeed a happy patient. There was recurrence of the fluid in the right ear two weeks later and the fluid was again aspirated. The left ear to be incised on two later occasions in a period of three weeks, but we are glad to state that this patient now has no further trouble and hearing has returned to its normal status. This is another case of incorrect therapy, which, finally, after four years' lapse of time was corrected. This case demonstrates that we usually find no damage to the acuity of hearing, or even the clarity of hearing, following secretory otitis media over a considerable period of time—in this particular instance, four years—if the fluid is finally removed.

CASE 3.—Mrs. M. F., age 52, April 1941. She gave a history of typical secretory otitis media, which she thought had existed in both ears, but which existed only in the right ear at the time of examination. Our examination revealed her statement correct. The right ear drum was incised and the fluid aspirated, with immediate restoration of hearing and relief of all symptoms. There has been

no recurrence of her symptoms. This was the first case of secretory otitis media wherein our treatment was employed—the incision of the ear drum and aspiration of the fluid. The aspiration was necessary when we observed that no fluid exuded from the myringotomy opening, although the fluid was very visible behind the drum. Since she had such complete relief of all of her symptoms, this treatment was decided on for all patients in the future with such symptoms and findings. This is a case of spontaneous secretory otitis media, since there had been no previous illness or other known cause of the disease.

CASE 4. D. H., age 48, October 1943. He had secretory otitis media with fluid level of the right ear. There had been mild allergy symptoms in the past and there was some hyperplastic lymphoid tissue in the nasopharynx. He had acute bronchitis at the time of the fluid in the ear. There was complete relief of all symptoms after myringotomy and aspiration of the fluid, but there was recurrence of the fluid in the right ear on three occasions. Radium therapy over the nasopharynx had been applied elsewhere during the duration of the disease. The right ear had barely cleared when typical symptoms developed in the left ear. A fluid level was found and the fluid was aspirated following myringotomy. As occurred on the right side, there was recurrence of the fluid on three occasions, each time the fluid being aspirated. There have been no further symptoms to date and hearing has been normal. The disease in this case could possibly be traced to allergy or hyperplastic lymphoid tissue, or both.

CASE 5.—P. D., age 6, January 1944. She had influenza one month previous to onset of symptoms. Fluid was found filling the right middle ear and there was a small amount of fluid in the left ear. Both drums were incised and the fluid aspirated. She was then well for a period of three months. Then, rather suddenly, she again developed fluid in both ears. The fluid was again aspirated and there has been no recurrence. A short time after the latter aspirations the recurrent adenoids were removed. This was an unusually cooperative child and, as usual, no anesthesia locally of any type was used for the myringotomies. One might conclude that this case demonstrates the occurrence of fluid in the middle ear following influenza, or even associated with the hypertrophied adenoids. (The tonsils had previously been removed.)

CASE 6.—Miss J. G., age 27, October 1945. The history showed sudden impairment of hearing with other symptoms of secretory otitis media involving the right ear for a period of ten days. The otolaryngological examination was entirely negative otherwise. The

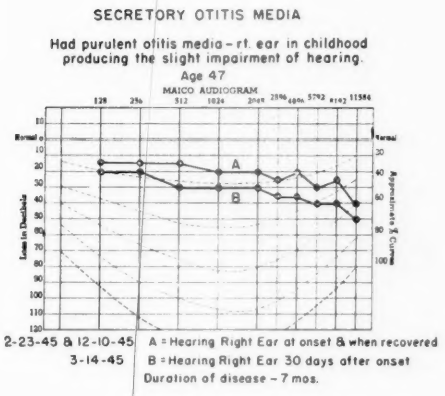


Fig. 4.—Audiogram of Case 7.

right ear drum was incised. The fluid was so thick that it could not be aspirated through the incision made. A counter-incision was then made anteriorly in the ear drum in order that air could be drawn through the middle ear. This is an example of the extremely tenacious and thick fluid which does not aspirate well and always requires a second incision in the ear drum for its complete removal. This also is another case of spontaneous secretory otitis media.

CASE 7.—Mrs. L. V., age 43, January 1945. The history was typical of secretory otitis media involving the right ear only. Examinations on several occasions failed to reveal any fluid in the middle ear until one week following the first examination, when a fluid level was found. The ear drum was incised and the fluid aspirated. In this case it was necessary to incise the drum and aspirate the fluid anywhere from one to five times weekly for a period of seven months until the secretion had completely ceased. During the interim she had x-ray therapy over the middle ear and mastoid process and nasopharyngeal lymphoid tissues. She went to the beach for a short period of time and she followed various other directions designated. There is a mild allergic history in this case, but otolaryngological examination was otherwise negative. Roentgenograms of the mastoid showed no disease. After a period of several months she is now free of the symptoms of secretory otitis media but does have some tinnitus, which may be attributed to hypertension (blood pressure 175/110). An audiogram shows return of her hearing to the pre-

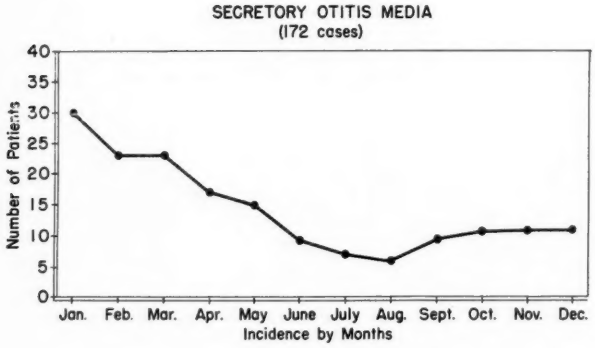


Fig. 5.

existing level in the right ear (Fig. 4). This case demonstrates the recurrent or persistent disease for a period of seven months with no apparent cause and no obvious reason for the continuation of symptoms, regardless of therapy employed, including catheterizations and inflations of the eustachian tubes, which we also employed while treating her.

CASE 8.—W. P., age 63, December 1943. There had been impaired hearing in the left ear all of his life due to an old inactive otitis media, but this ear had been dry since childhood. There had never been any symptoms in the right ear. For many years he had suffered from asthma and other allergic phenomena. One week previous to examination he had rather sudden onset of typical symptoms of secretory otitis media in the right ear. After visualization of fluid, the drum was incised and the fluid aspirated. This treatment of myringotomy and aspiration was found necessary at very frequent intervals throughout a period of eleven months before the secretion finally ceased. During those eleven months he had numerous catheterizations and inflations, although he was always able to inflate the middle ear by the Valsalva method. Roentgenograms of the right mastoid showed no abnormality or infection. He has now been free of all symptoms for more than one year and the ear drum, in spite of all the myringotomies, appears fairly normal and hearing is back to the original level. This is the most prolonged case that has come under our observation, and it seems to demonstrate that catheterizations and inflations, even combined with myringotomy on

many occasions, do not clear up the disease, and such a method of treatment through trauma, although mild, may be a factor in prolonging the condition.

SUMMARY

A disease formerly known as "catarrhal otitis media" but now more aptly termed "secretory otitis media" has been discussed, and because of the increased frequency of the disease in the past four years, this discussion is considered very timely. The symptoms, the physical findings, the treatment and prognosis of secretory otitis media have been outlined. A group of 172 patients, all except one having been seen in the past four years, has been analyzed. Of these, eight cases were chosen for presentation because of the various phases of the disease which they presented.

The newer method of treating secretory otitis media—that of incising the ear drum and aspirating the fluid—has shown excellent results, and we consider this method the only correct one for treatment of patients with the disease. Complications are practically nil. Of the 172 patients only one developed purulent otitis media following myringotomy and aspiration of the nonpurulent fluid.

We have offered no definite causative factor for the increased frequency of the disease, although we know, as did our predecessors of 70 or 80 years ago, that a eustachian tube blockage is the primary cause of secretory otitis media. There is yet no evident cause of the eustachian tube blockage in most of the cases. *It is possible that we are dealing with a virus disease;* since the disease is almost in epidemic form, there is reasonable ground for this suspicion. We have been unable to correlate the use of sulfonamides in recent years with the disease, since they were used for almost five years prior to the so-called epidemic of secretory otitis media (Fig. 5). We do admit that secretory otitis media is more prevalent in the cold months of the year, but we do not admit that the disease is necessarily associated with upper respiratory infections. We are reluctant to admit that the disease had been prevalent in the years prior to 1941 and that our eyes are now seeing pathology which they had previously failed to see.

DUKE HOSPITAL.

VI

WAR DEAFNESS IN GUNNERY INSTRUCTORS

CAPTAIN JOHN E. HENDRICKS

MEDICAL CORPS, ARMY OF THE UNITED STATES

Impaired hearing and tinnitus, due to gunfire and high explosives, was the subject of many papers during and immediately following World War I. For the next 20 years, however, the relatively few publications that appeared dealt for the most part with acoustic trauma in industry.

During the present war many men in army training and in combat are subjected to the excessive noise of gunfire and explosives, and during the past few years many publications have appeared on this subject.

At an Army Air Force Flexible Gunner School* in Florida our attention was focused on this problem by the almost daily appearance at the ear clinic of students with the complaint of deafness or tinnitus. Usually the onset was sudden, coming on after a single day's firing. Others described a cumulative effect, feeling that each additional day's exposure increased their symptoms. The latter group frequently dated the onset of the symptoms from the first day on the ranges. Some had had no trouble with their ears until they were subjected to an unusually loud nearby explosion. The .45 caliber pistol, the .50 caliber machine gun, and the 20-gauge shotgun appeared to be the worst offenders. Smaller caliber guns seldom caused trouble. Many students had no ear symptoms and consequently were never examined. Students would have offered excellent material for a study of the various stages in the development of acoustic trauma from gunfire had it been possible to make audiograms on all as soon as they arrived at the field and at frequent intervals during their training. This procedure was not practical because their period of training was too short for adequate observation. Instead, the gunnery instructors were selected for this study.

Fifty-two men selected at random from the instructor's squadron were observed for an average of 123 days. No records were available of their hearing acuity at the time of their entrance into the service, but 48 stated that their hearing was normal and 4 admitted

*Tyndall Field.

mild monaural deafness at the time of induction. Their length of service as instructors varied from 7 to 720 days with an average length of 169 days, but prior to being assigned as an instructor each man had had from six to ten weeks intensive training on the firing range. In other words all had been subjected to some aural trauma before this study was begun.

A complete ear, nose, and throat history and examination, including audiograms, was made at the beginning of the study on each of the 52 instructors and repeat audiograms were made at intervals during the period of observation. From three to four audiometric tests were made on each man. All tests were conducted in a quiet, but not soundproofed room of cement and tile-stucco construction. The Maico audiometer used was checked for accuracy by repeated audiograms on the same normal ears. In 71 per cent, the initial tests were made within three hours of the time the subjects left the ranges and 82 per cent of the final tests were also made within three hours of working on the ranges.

Because the hearing acuity of this group of instructors prior to their first exposure to machine guns is unknown, we have used the audiograms of a "normal group" reported by Senturia[1] as controls. Senturia's group consisted of 500 aviation cadets of an average age of 21.1 years who were tested at least 30 days after exposure to excessive noise. The average age of the men in our study was 23 years and they were physically quite comparable to Senturia's cadets.

The system of classifying audiograms devised by Senturia is used in the present report. Briefly, the threshold audiometric curves are divided into four types: Type A—showing no more than a 10 decibel variation from the accepted normal in the frequency range 1,024 to 11,584 cycles per second; Type V—having a typical and clearcut V-notching or tonal dip; Type W—having in addition to the V-notch a loss of more than 10 decibels in the higher or lower frequencies; Type T—characterized by a progressive or variable high tone loss, greater than 10 decibels. To record the severity of a loss, Senturia graded each threshold curve numerically for the greatest loss for any frequency above 1,024 cycles per second. With reference to the zero line, Grade 0—plus or minus 10 decibel change in the curve; Grade 1—losses of 15 to 20 decibels; Grade 2—losses of 25 to 40 decibels; Grade 3—losses of 45 to 60 decibels; and Grade 4—losses greater than 60 decibels.

When the audiograms of the 500 average normal cadets of Senturia are examined as to type of hearing losses, 33 per cent of the ears

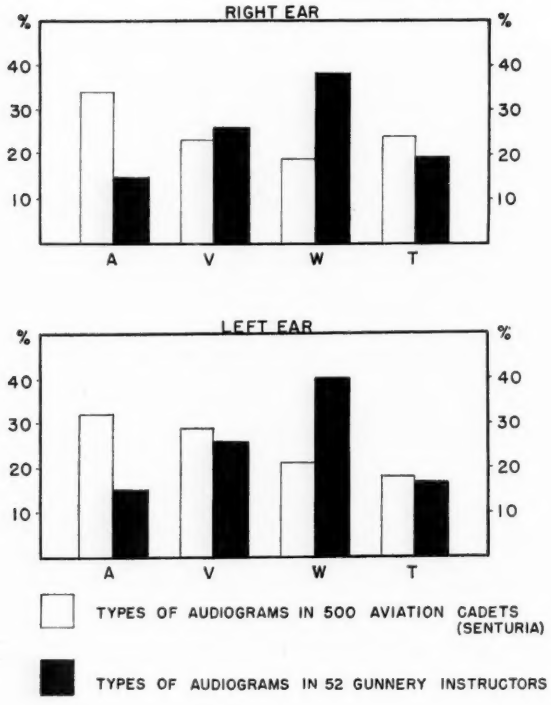


Fig. 1.

show threshold curves that deviate not more than 10 decibels below the accepted zero line for any frequency from 1,024 to 11,584 cycles per second (Type A) and Type V occurred in 26 per cent of the ears. In contrast the initial audiograms of the gunnery instructors have greater numbers of Type V and Type W curves (Fig. 1). When the audiograms of these 500 controls are analyzed as to grades of deafness, the majority are found to be Grade 0 or Grade 1. In contrast, the majority of the initial audiograms of the gunnery instructors fall into Grade 2, Grade 3, and Grade 4 (Fig. 2). These facts indicate that these 52 instructors at the time their audiograms were made, that is, after an average of 169 days on the firing line, have suffered extensive damage to their auditory apparatus.

The Weber and Rinne tests were made on only 44 of the instructors at the initial examination. The 512 d. v. steel fork was used. The

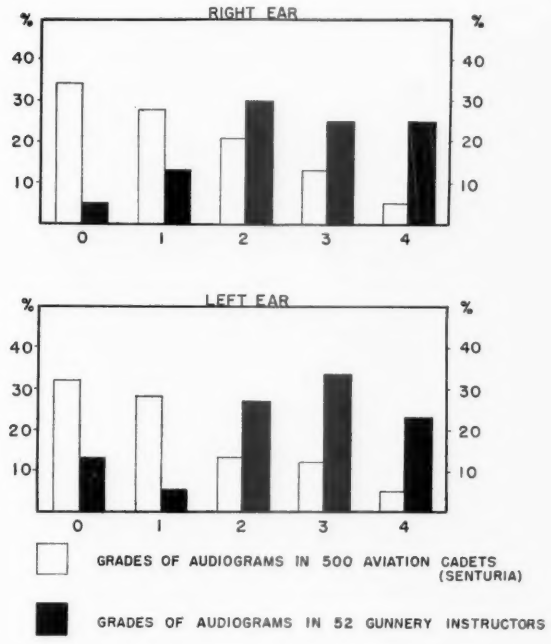


Fig. 2.

Rinne test indicated shortened bone conduction for 18 men, and in the Weber test, the sound was lateralized to the left ear in 11 and to the right ear in 7 men. The types and grades of audiometric curves of these 36 men differed in no way from the average for the entire group.

Every instructor in this study worked on the machine gun ranges. These ranges were on a sandy beach with guns mounted singly six feet apart, or mounted in pairs in turrets on two-and-one-half ton trucks, lined up hub to hub. There were from 16 to 20 guns on each range; half of the guns used were .30 caliber and the other half were .50 caliber. The men were on duty eight hours a day and the guns were actually fired about six hours a day. The firing was done in bursts of about 12 shots each. All guns were individually operated, and these short ripping bursts might come sporadically or all together. Each instructor stood directly behind his student to guide and help him.

The "peak crash" or pitch of the noise produced by a single .50 caliber machine gun has been estimated to exceed 10,000 cycles per second, and the intensity of the noise to exceed 170 decibels.[2] Usually each instructor was off duty one day each week and frequently they were given furloughs, or three day passes. At one period covered by these tests they worked on the firing lines in the evenings as well. During this period the complaints of tinnitus and deafness increased temporarily.

Forty-six of the 52 instructors wore cotton constantly. Two used it only occasionally and four used nothing, because they either could not tolerate the cotton or could not converse easily with their students. No other protection was used.

The following table shows the pertinent findings obtained from the history records:

TABLE 1.—HISTORY OF 52 GUNNERY INSTRUCTORS

Tinnitus at initial examination	27
Tinnitus at final examination	30
Deafness at initial examination	15
Deafness at final examination	21
Good hearing at time of induction	48
Poor hearing in one ear at time of induction	4
History of deafness in family (parents)	6
History of deafness in family (others)	7
Past history of otitis media	9
Occupied in noisy civilian job longer than one year	6
Occupied in noisy civilian job less than one year	7
Experienced in hunting (more than one season)	25

An accurate history as to time of onset of the tinnitus or deafness was difficult to obtain. The appearance of symptoms was insidious and varied from the first day on the range to the fourteenth month. Surprisingly, only a few of the men complained of constant tinnitus. Most of them stated that tinnitus ordinarily was present throughout the evening after a day on the ranges, but had disappeared by the following morning. This was true even in those with rather severe high tone losses. In contrast to tinnitus the men who complained of impaired hearing were constantly and often acutely aware of it. Nearly all felt their impairment was increased for an hour or two after each day's work.

The number with a family history of deafness among the men in this series was too small to attempt a correlation of susceptibility to acoustic trauma and a familial tendency to deafness.

A history of otitis media in childhood was obtained from nine men. One had had a mastoidectomy. Two of these nine men were

among the four who admitted poor hearing at the time of induction. There was nothing in this group to indicate that otitis media in the past had increased the susceptibility to deafness from gunfire.

Twenty-one men at one time or another had experienced pain in their ears after unusually loud explosions. The usual description was that it felt like an ice pick had been jabbed into their ears, but the pain was only momentary. No case of ruptured tympanic membrane was observed.

Table 2 presents the pertinent ear, nose, and throat findings obtained at the original examination:

TABLE 2.—RESULTS OF EAR, NOSE, AND THROAT EXAMINATIONS ON 52 GUNNERY INSTRUCTORS.

Scarring of membranae Tympani	15
Retraction of membranae tympani	9
Abnormal eustachian tube orifices	7
Fossa of Rosenmüller filled with lymphoid tissue	23
Adenoids: Large mass of lymphoid tissue	19
Small amount of lymphoid tissue	28
No lymphoid tissue	5
Tonsils present	32
Infected lymphoid tissue on posterior pharyngeal wall	5
Cloudy antra (or other evidence of sinusitis)	13
Nasal polyps	1

Of the 15 men presenting scarred tympanic membranes only four had a definite past history of otitis media, and only four had retracted drumheads. The other findings are given merely to complete the picture and have little relationship to the problem of acoustic trauma. One of the men with a slit-like eustachian orifice, hyperplastic lymphoid tissue in his nasopharynx, and mildly scarred tympanic membranes was one of the four who had a history of moderate hearing impairment at the time of his induction.

Effect of Prolonged Gunfire on Hearing. After establishing the degree of hearing impairment in the instructors at the time the study was begun, observations were made on subsequent changes appearing in the audiograms of the same men during an additional 123 days' (average) exposure to gunfire.

The effect of the prolonged exposure to gunfire in this group was manifested by a progressive loss of hearing in 45 men (86.4 per cent of the group). A composite of the air conduction losses for each frequency for each ear was made from the initial audiograms, and another from the final audiograms of the 52 men (Fig. 3). There

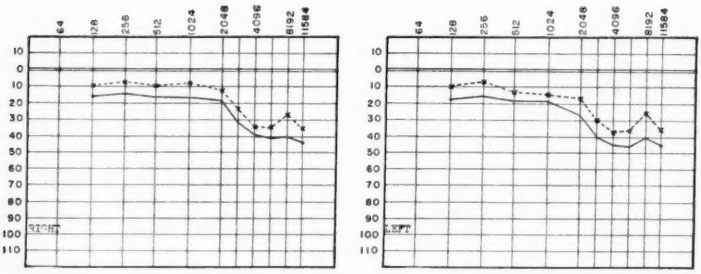


Fig. 3.—Composite audiograms of the air conduction of 52 men.
..... Audiogram at time of initial examination (average 169 days' exposure).
——— Audiogram at time of final examination (average 123 additional days' exposure).

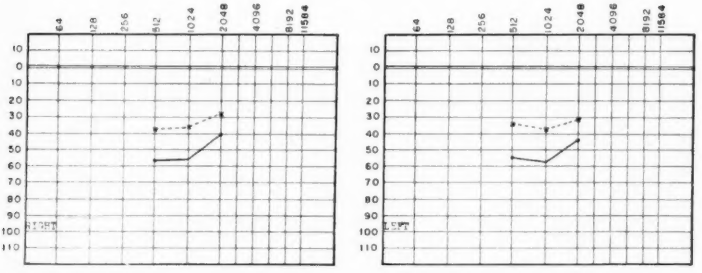


Fig. 4.—Composite audiograms of the bone conduction of 52 men.
..... Audiogram at time of initial examination (average 169 days' exposure).
——— Audiogram at time of final examination (average 123 additional days' exposure).

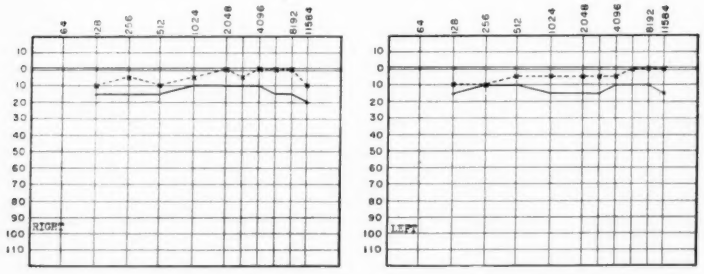


Fig. 5.—Example of undamaged hearing after many months' exposure to twenty .50 and .30 caliber machine guns.
..... 9 months' exposure (2½ hours after firing).
——— 13½ months' exposure (2 hours after firing).

was a 5 to 13 decibel loss in each ear for all frequencies after 123 days' (average) additional exposure to gunfire. The greatest change occurred in the 8192 frequency for both ears. The same procedure was worked out for the bone conduction losses for the three central frequencies, and a 10 to 20 decibel loss was noted after 123 days' additional exposure to gunfire (Fig. 4). A 5 to 10 decibel variation in audiometric thresholds is considered within normal limits, but the changes in Figs. 3 and 4 are an average of the entire group and therefore must be considered significant.

A study of all of the ears revealed that the left ear was more severely involved in 26 men, the right in 14, and in 12 both the right and the left ear were equally affected. No explanation is offered for this except that perhaps the instructor's left side was habitually nearest the muzzles of the guns.

Progressive impairment of hearing as a result of exposure to gunfire is apparent when the audiograms are analyzed in respect to the length of service on the firing ranges. During the period of this study 364 tests were made on 104 ears and the audiograms classified according to type and grade. They were then distributed according to the number of months' service each man had on the ranges, as shown in Tables 3, 4, and 5. In Table 3 the percentage of Type T audiograms increases with each additional month's exposure. In Table 4 the Type A hearing has a tendency to progress to Type V, W, or T. The Type V deafness progresses to Type W and Type T, the Type W to Type T, and Type T remains as it is. All of the types in this table tend to progress toward the more severe hearing defects (except three which show improvement). Similarly, when the audiograms are classified according to grade (Table 5) more severe losses are evident as additional time is spent near the guns.

TABLE 3.—EFFECT OF ADDITIONAL MONTHS EXPOSURE TO GUNFIRE ON THE TYPE OF HEARING CURVE.

MONTHS	1	2	3	4-6	7-9	10-12	13-18	19-24	25-30
NUMBER OF AUDIOGRAMS	20	20	22	122	82	42	38	10	8
			(Figures below are in percent)						
Type A	5	5	9	13	10	5	5	20	12
Type V	25	30	14	16	5	2	3	0	0
Type W	40	35	23	28	26	17	13	0	0
Type T	30	30	54	43	59	76	79	80	88

Individuals differ in their ability to withstand severe noise. As mentioned before some students complained of tinnitus after the first burst of firing and others never complained. This was true of the

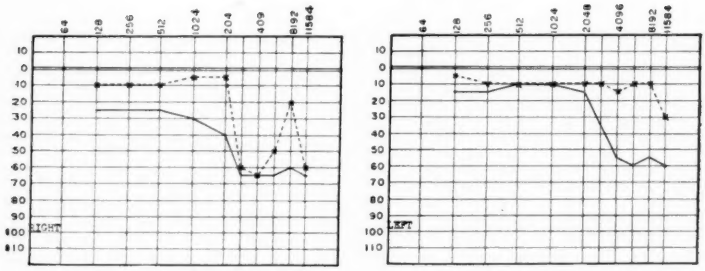


Fig. 6.—Example of damaged hearing after relatively short exposure to twenty .50 caliber machine guns.
..... ½ month's exposure (27 hours after firing).
——— 4½ months' exposure (3 hours after firing).

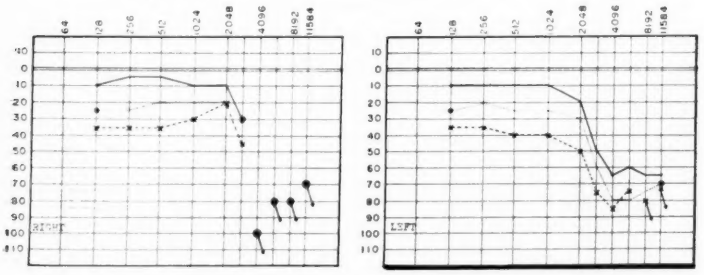


Fig. 7.—Three audiograms made on the same individual after varying number of days' rest from gunfire. Marked recovery takes place for certain frequencies, but in others no improvement is noted. Original exposure was to 62 (caliber .50) machine guns (malfunction range).
..... 16 months' exposure (2½ days after firing).
..... 16 months' exposure (30 days after firing).
——— 16 months' exposure (80 days after firing).

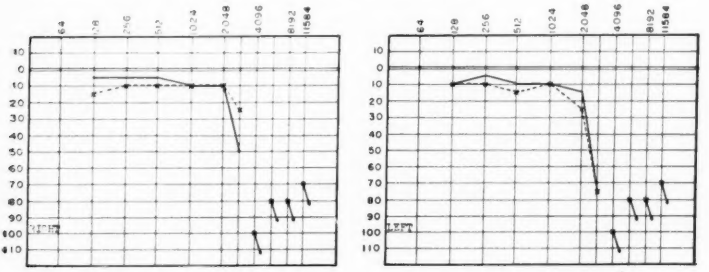


Fig. 8.—Example of severe hearing impairment following 11 months' exposure to machine gun fire showing little change after a rest of six weeks.
..... 11 months' exposure (3 hours after firing).
——— 11 months' exposure (6 weeks after firing).

instructors also; several ears were damaged early in the exposure period while others remained normal for months (Figs. 5 and 6).

TABLE 4.—PROGRESSION FROM MILD TO MORE SEVERE TYPE DEAFNESS WITH ADDITIONAL EXPOSURE TO GUNFIRE.

104 Ears at Initial Test.	Same 104 Ears 123 Days Later	
ORIGINAL TYPES	UNCHANGED	PROGRESSION TO MORE SEVERE TYPES
16 Type A	11 Type A	plus 2 V, 2 W, and 1 T.
29 Type V	11 Type V	plus 8 W, 10 T.
40 Type W	23 Type W	plus 15 T, 2 reverted to V.
19 Type T	18 Type T	plus 1 reverted to A.

TABLE 5.—EFFECT OF ADDITIONAL MONTHS TO GUNFIRE ON GRADES OF HEARING LOSSES.

MONTHS	1	2	3	4-6	7-9	10-12	13-18	19-24	25-30
NUMBER OF AUDIOGRAMS	20	20	22	122	82	42	38	10	8
			(Figures below in percent)						
Grade 0	0	0	10	6	3	2	0	10	0
Grade 1	20	5	5	12	7	8	8	10	12
Grade 2	30	35	26	27	28	12	5	10	12
Grade 3	30	40	33	32	41	26	32	20	26
Grade 4	20	20	26	23	21	52	55	50	50

It seems probable that ears subjected to excessive noise suffer a marked hearing loss early, and after that continue to lose only gradually. One can assume from this and from observations of the frequency most affected that the cochlear end organs in the basal turn are easily injured by acoustic trauma, while those in other turns are less affected. This hypothesis is supported by observations made immediately and those made soon after several weeks or months of exposure to severe acoustic trauma. The damage appears to be permanent for the higher frequencies, while the lower frequencies usually show some recovery after rest (Fig. 7).

Effect of Rest Upon Hearing Damaged by Acoustic Trauma. It was expected that ears with marked hearing impairment would show considerable improvement after adequate rest. The amount of improvement in most cases was disappointing and while a few showed considerable recovery (Fig. 7), others showed very little (Fig. 8). Seven men, whose experience as instructors averaged over eight months, were tested immediately before a furlough and again after their return fifteen days later (Fig. 9). The averages of their audio-

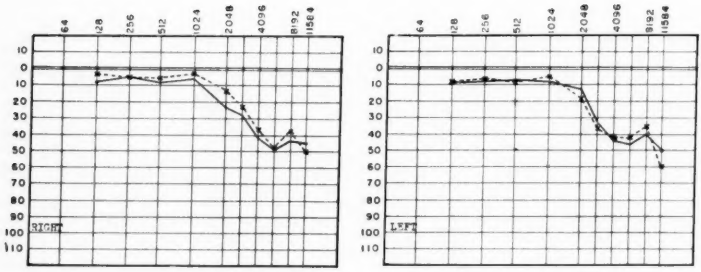


Fig. 9.—Composite audiometric curves of seven men with an average of 259 days' exposure to machine gun fire immediately before and after a 15 day furlough. Example of little improvement after rest.
..... 8½ months' exposure (3 hours after firing).
——— 8½ months' exposure (15 days after firing).

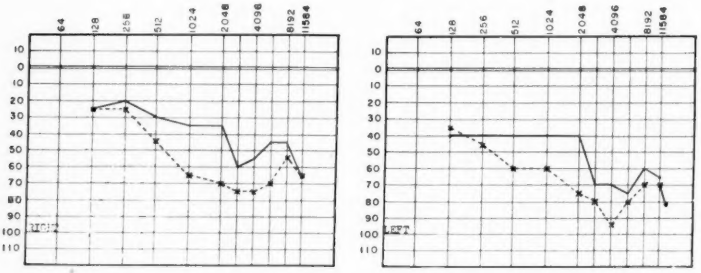


Fig. 10.—Partial recovery of hearing in practically all the frequencies which had been severely damaged by six weeks' exposure to gunfire.
..... 6 weeks' exposure (6 hours after firing).
——— 6 weeks' exposure (30 days after firing).

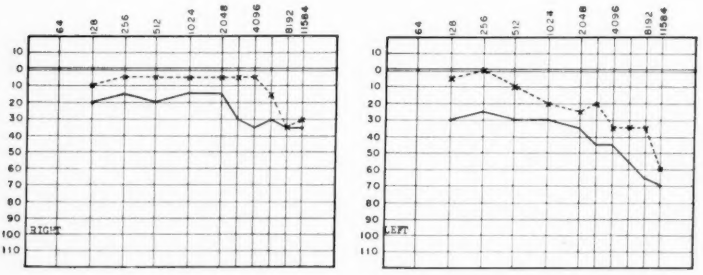


Fig. 11.—Example of hearing becoming worse with two months' additional firing followed by four months' rest from the machine gun ranges.
..... 7 month's exposure (3 hours after firing).
——— 9 months' exposure (4 months after firing).

grams show no significant improvement. On the contrary, there is a slight loss. These observations indicate that impairment tends to remain permanent rather than to recover.

Four men who had severe hearing losses were removed from their positions on the firing line and observed to see how much recovery took place. One had but little change in the damaged hearing after six weeks of rest (Fig. 8); in another the hearing improved markedly for some tones after 2½ months of rest (Fig. 7), and one had a partial recovery for practically all frequencies after thirty days (Fig. 10). The instructor whose record is shown in Fig. 11 worked on the ranges for an additional two months after the first test, then had a four month rest. In spite of this rest his hearing impairment progressed.

Effect of Age Upon Hearing Impairment Due to Acoustic Trauma. Age apparently plays some part in the susceptibility to cochlear drainage. In this series the ages varied from 18 to 33 years. Only 13 men were 25 years old or more. The group is too small to determine with accuracy the effect of age, but the following table seems significant.

TABLE 6.—CLASSIFICATION OF 104 INITIAL AUDIOGRAMS ACCORDING TO AGE

AGE GROUP	NUMBER OF MEN	NUMBER OF EARS	TYPE A	TYPE V	TYPE W	TYPE T
18-24	39	78	16%	23%	35%	24%
25-33	13	26	3%	15%	38%	42%

In Table 6 the younger group shows a more even distribution of the various types of audiograms, whereas in the older group the W and T types predominate.

Plans for observations on the effectiveness of preventive measures were made but were interrupted by transfer of the author to other duties at another station.

SUMMARY

1. Fifty-two machine gun instructors from an Air Force Flexible Gunnery School were observed for hearing losses over a period averaging 123 days. An average of 3.8 audiograms was made for each man.

2. Forty-five men (86.4 per cent of the group) had an increase in hearing impairment during the period of observation. The losses in hearing involved the entire scale. The average loss was greatest

for the frequency of 8,192 cycles per second (13 decibels in the left and 12 decibels in the right).

3. The degree of impairment generally progressed in accordance with the length of exposure, but some individuals resisted cochlear damage for months while others suffered severe losses relatively early.

4. Seven men were tested immediaely before and after a 15 day furlough. No definite recovery of hearing losses was noted from a composite of their audiograms. Four other men were re-examined after 30 to 120 days absence from the firing lines. The recovery was disappointing. Generally the lower frequencies recovered more than the higher.

14 West Locust St.
Newark, Ohio.

REFERENCES

1. Senturia, B. H.: Auditory Acuity of Aviation Cadets, Annals of Otology, Rhinology and Laryngology 53:705 (Dec.) 1944.

2. Kos, C. M.: Personal communication, 1944.

VII

PSYCHOGENIC DEAFNESS

Lt. Col. Norvil A. Martin

Medical Corps,

Army of the United States

During the past two years more than 1500 patients have been admitted for deafness to the Hearing Center at Hoff General Hospital, Santa Barbara, California. Careful records have been kept of the various types of deafness presented, resulting in a recent study and evaluation of cases based upon etiologic factors. Although most of the cases of deafness studied can be attributed to organic disease, a large number of patients were found to have deafness on a psychogenic basis. It is with this factor that the present paper is concerned.

Psychogenic deafness is not new. Otologists have long recognized the problems that are encountered in the diagnosis and treatment of such cases. However, with the recent emphasis on narcosynthesis in war neuroses as presented by Colonel Roy R. Grinker in his report on such cases in the United States Army in Africa, and with the difficulties encountered in evaluating many cases of deafness on a truly organic basis, it was deemed advisable to study selected cases under sodium pentothal narcosis. The first cases gave such startling results that 75 cases have been selected for study out of the last 500 patients with deafness admitted to this hospital for aural rehabilitation. Under the term "psychogenic deafness" will be included all the various types of neuroses, and no attempt will be made in this otological presentation to classify or study cases from the psychiatric point of view. Such a report has already been presented in a paper by Rosenberger and Moore[3] who have dealt with the treatment of hysterical deafness as found at this hospital.

Each patient who is deaf has a complete hospital work-up and two audiometric tests on successive days prior to a complete otological examination. A psychiatric consultation follows by the Neuropsychiatric Section and a psychological evaluation is made by the clinical psychologist. The problem of establishing the criteria for the selection of cases to be treated under sodium pentothal narcosis has been an interesting one. The otologist has the best opportunity to select these since he can determine whether ear pathology is present.

A positive finding does not, however, preclude the possibility of the existence of a functional overlay. Thus, in all cases there must be a complete evaluation of the significant psychological factors in order to determine the need for narcosynthesis. The combined opinion of all examiners, including the otologist, the psychiatrist, and the psychologist, is essential for the best selection of cases. Many other cases, I feel, may be benefited by narcosynthesis as a form of therapy, but since it is not practical to use this type of examination and treatment on all cases of deafness, it is employed only with selected individuals.

A positive finding that has been prominent in selecting such cases has been a history of deafness that does not coincide with the usual clinical history in judging deafness from the pathological point of view. Other indications include: repeated audiometric tests that do not fall within the normal expected degree of variation; responses to the low conversational voice and to the whispered voice that do not conform; and labyrinthine tests that show no dysfunction. In these cases there is often great variation between the binaural audiogram administered in the "free field" room and the usual monaural audiograms, and results on the recorded speech perception test are inaccurate. No improvement or a startling increase in hearing with a hearing aid may be a positive indication. In these cases there is usually bilateral profound loss of hearing to the pure tone audiometer and to speech perception tests. Various malingering tests can also be done but they help very little in determining the level of hearing in psychogenic deafness. No one or two of these tests may be sufficient for adequate diagnosis. The patient's ability to hear should be evaluated only after a thorough study for ear pathology and after gathering complete data on all possible hearing tests. In the table that follows it is significant to note that 35 of the "noncombat" men had deafness noted prior to military service.

The classification of cases that we have used at this hospital conforms very closely with the classification in a recent article by Zilboorg[1], who reports the classification by E. M. Zalkind.[2] In the latter study, the cases which were discussed were examined almost immediately following injury in combat, whereas our combat patients did not arrive at the Hearing Center until from one to three months after they had left the active area. Our observations, however, closely parallel what the Soviet Army has found in its use of light narcosis in treatment of psychogenic deafness.

The classification that we use in describing psychosomatic deafness is as follows:

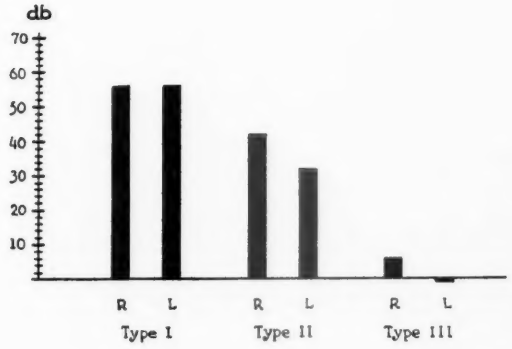


Fig. 1.—Average improvement in decibels within the speech range (256-2048 d.v.)

TABLE 1.

	NO. OF CASES	% IN 500 CASES	% IN 75 CASES STUDIED	AVERAGE AGE	MOS. OF SERVICE
I. Psychogenic deafness with no or slight organic loss				26	26
Combat	4	0.80%	5.33%		
Noncombat	22	4.40%	29.33%		
	26	5.20%	34.66%		
II. Psychogenic deafness with organic disease				33	27
Combat	9	1.80%	12.00%		
Noncombat	26	5.20%	34.66%		
	35	7.00%	46.66%		
III. Unimproved cases				32	21
Combat	4	0.80%	5.33%		
Noncombat	10	2.00%	13.33%		
	14	2.80%	18.66%		

This table is based on 75 cases studied out of 500.
Note: The average age of 500 patients is 29 years.

Type I Psychogenic deafness with no or slight* organic loss.

Type II Psychogenic deafness with organic disease.

Type III Unimproved or control cases.

Patients selected for study as possibly having psychogenic deafness equaled 15% of all admissions. Twelve per cent of all patients admitted had complete return of hearing to normal or a substantial improvement.

The total number of blast injuries in 500 cases studied numbered 36. By blast injury it is inferred that the onset of deafness occurred following one single incident of acoustic trauma under combat conditions. Forty-seven per cent of patients with deafness associated with a history of blast injury in combat were chosen for narcosynthesis under the criteria as previously given in this paper. Thirteen of 17 patients with such a history showed pure psychogenic deafness or organic deafness with a functional overlay. It is also of interest that our experience with these patients with acoustic trauma chosen for psychological care conforms closely with the experiences in the Soviet Union as stated by Zilboorg[1] in which 50 per cent of the deafness acquired under combat conditions was discovered to be on a psychological basis. We have had, however, no cases of deaf-mutism admitted for study, although one developed a functional aphonia lasting for 48 hours after narcosis. In several cases there was also other functional manifestations such as corneal anesthesia, tubular vision, "glove" anesthesia of extremities, stuttering, and for a short time following narcosis, amnesia in two cases.

The treatment of these patients is carried on by a psychologist under the direct supervision of the Chief of the Neuropsychiatric Section. Intense suggestion is given to the patient and he is convinced in three or more interviews prior to narcosis that he will be able to hear normally. In the cases thought to be totally functional, the patient is introduced to other patients who have recovered their hearing through narcosis. He receives their assurance of success. Group suggestion is also carried out in the typical Army informal discussion groups, where patients about to be treated and those who have recovered are brought together to talk over their difficulties. When it is felt that the patient has been prepared psychologically for narcosis, he is given sodium pentothal. Deep narcosis is not necessary. If the patient is carried beyond light narcosis, therapy must stop until he is

*Slight loss refers to a slight high tone loss as demonstrated in the following case reports.

able to respond to questioning. The patient is instructed to count backwards from 100 to 1, and in this he usually becomes incoherent before he has counted to 50. The anesthetist gages the speed of his injection so that he is able to keep the patient in the so-called "babbling narcotic state." The patient is asked simple questions, and he is assured and reassured that he is now about to recover his hearing. The external ear is sprayed with ethyl chloride so that there is a definite temperature change and the patient is then assured that his hearing has returned. The therapist asks further questions; sometimes a great deal of suggestion and persuasion is required to secure answers. As the patient answers questions, one ear at a time is blocked and the other ear tested. The total procedure takes approximately 20 minutes, but the patient is observed continuously for the next one or two hours. This procedure may be prolonged where there is willful simulation of deafness. During this time he is continuously being assured that his hearing is normal until he is fully conscious and realizes that he is hearing. Some patients are given sodium amytal orally after sodium pentothal to prolong the narcotic state.

The patient then has audiometric, spoken and whispered voice tests recorded as soon as possible following narcosis. Pure tone audiometric tests are run by the same technician. This technician was specially trained by the psychologist in the proper manner of handling these men. These tests are repeated the following day, and as often thereafter as necessary to determine the threshold of hearing. The attention factor is very important in testing a patient who is slightly stuperous, and it is necessary for the psychologist to be with the patient and constantly call his attention to the fact that the tester is trying to get a response. Recorded speech perception tests are run as a further determination of the threshold of hearing if there is any question of the results of the live voice tests.

Emotional blocks and conflicts that have caused the symptoms of hysteria are carefully evaluated and approached psychologically in an effort to prevent the patient's symptoms from returning. In successful cases the patients relive their combat or noncombat experiences while under light narcosis, achieving a final relief of tension. Some patients, when they become conscious of the fact that they are hearing, will suddenly revert to their former state. It is felt that they are willfully simulating their symptoms.

The true medical evaluation of a patient's hearing is of utmost importance prior to any educational program. It is felt that it is distinctly harmful to admit psychogenically deaf patients to an

educational program before the psychological factors are fully understood.

Prior to the use of sodium pentothal in the treatment of such cases, ten hearing aids with 90 volt "B" battery units were dispensed. This type of battery arrangement is unusual and is resorted to only in cases of profound deafness where it provides great amplification. Since sodium pentothal has been used, it has not been found necessary to supply a single 90 volt unit.

CONCLUSIONS

1. Psychogenic deafness is not rare and an approach to its evaluation is greatly facilitated under sodium pentothal narcosis when it is carried out under careful and proper technique. Such an evaluation is important prior to an educational program for the care of deafness. This factor must also be considered in evaluating improvement obtained in medical treatment and educational programs, both under war time conditions and in civilian programs in the era of peace which is to follow.

2. Of the cases of deafness studied under sodium pentothal narcosis at Hoff Geneal Hospital, a significant group (12 per cent) were found to be pyschogenic deafness.

3. Thirty-eight per cent of all cases of blast deafness admitted to this hospital were found to be pure psychogenic deafness or organic deafness with a large functional factor.

4. Since the largest group of patients selected for study had deafness not associated with combat, the discovery of a significant psychogenic involvement in these patients offers a challenge for otologists in civilian practice.

5. For the careful selection of cases the complete cooperation of the otologist, the psychiatrist, and the psychologist is highly desirable.

6. Further study is necessary in the development of criteria for the selection of cases to be treated by narcosynthesis.

7. A simple test would be desirable for screening all cases of deafness for psychosomatic factors. This problem is being explored through the use of the conditioned reflex pathways to sound.

8. This article is not intended to convey the idea that narcosynthesis is a foolproof method of diagnosing all cases of psycho-

genic deafness. It has proved itself at this hospital, however, as a highly useful procedure, yielding much information not obtainable by accepted otological methods of examination.

Acknowledgement is extended to Major S. N. Palevsky, MC, Chief of Ear, Nose and Throat Clinic, Lieutenant Colonel A. I. Rosenberger, MC, Chief of Neuropsychiatric Section; Captain James H. Moore, AC, Clinical Psychologist; and Lieutenant M. Bergman, MA, Acoustic Officer, for their help in gathering statistics for this report. Major Herman Semenov, MC, has reviewed these cases and offered valuable suggestions in the preparation of this report.

Appreciation is also extended to Lieutenant Colonel James R. Patrick, AGD, on his suggestion to study the conditioned reflex pathway to sound.

THE GALLIPOLIS CLINIC
GALLIPOLIS, OHIO.

REFERENCES

1. Zilboorg, Gregory: Some Aspects of Psychiatry in the U.S.S.R., Amer. Rev. of Soviet Med., August, 1944.

2. Zalkind, E. M.: Neuropathology and Psychiatry 12:18, 1943.

3. Rosenberger, A. I., and Moore, J. H.: The Treatment of Hysterical Deafness at Hoff General Hospital, Santa Barbara, California, July 1945. (Unpublished)

4. Atkinson, Miles: Tr. Amer. Otol. Soc., 1937.

5. War Department Technical Bulletin: Treatment Program for Psychiatric Patients in Station and General Hospital, 1 April 1944, Par. 5b.

6. War Department Technical Bulletin: Treatment Program for Psychiatric Patients in Station and General Hospital, 10 August 1944, Par. 5 (2) c.

7. War Department Technical Bulletin: Neuropsychiatry for General Medical Officer, 21 September 1944, Par. 11, c and d.

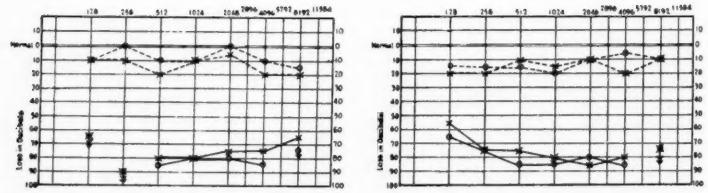


Fig. 2.—Patient, 28 years old, 3 years and 5 months in service. He has been hard of hearing for the past two years following a bomb explosion. He had purulent otitis media in both ears in 1942, and notices occasional tinnitus. There was marked hearing tension in this psychologically unstable individual. He had normal ears with purely psychogenic deafness.

Fig. 3.—Patient, 26 years old, 2 years and 3 months in Army service. A shell landed near this patient in November 1944 in combat, followed by deafness. He had bleeding from the right ear and complained of tinnitus. This patient had considerable combat reaction in the nature of a war neurosis. He had normal ears with purely psychogenic deafness.

Note: In these audiograms X indicates the left, O the right ear; continuous lines, before narcosis; broken lines, after narcosis.

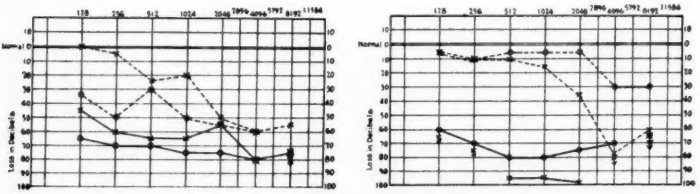


Fig. 4.—Patient, 35 years old, 22 months in the Army. He has been deaf since January 1943 without cause. Hearing to whispered voice and spoken voice were consistent, but the audiograms were low. Following narcosynthesis the whispered voice and spoken voice remained constant and the audiograms improved. Organic disease with psychologic factors was present. Actual proceedings of psychotherapy have been electrically transcribed on this case.

Fig. 5.—Patient, 26 years old, 34 months in the Army. He has been hard of hearing for 13 years with marked psychological tension built around his occupation as a railroad switch tender. There is psychogenic deafness. Actual proceedings of psychotherapy have been electrically transcribed on this case.

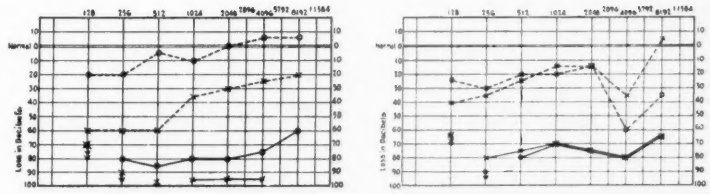


Fig. 6.—Patient, 20 years old, 4 years and 2 months in service. An exploding shell in combat caused bleeding from the left ear. A marked anxiety state is present. There is a central perforation of the left ear drum. The patient has organic disease with a marked functional overlay.

Fig. 7.—Patient, 25 years old, 3 years and 9 months in service. This patient was exposed to 155 mm. firing and had bled from the ears from concussion blast. There is mild hearing tension. He has organic disease plus a functional overlay.

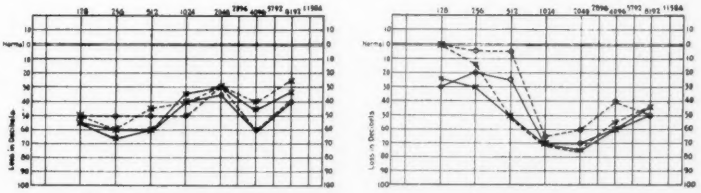


Fig. 8.—Patient, 24 years old, 9 months in the Army. His deafness has been of two years' duration, progressive in nature, and there is a family history of deafness. No hearing tension was noted. Following narcosis, there was no improvement. Organic disease only is present.

Fig. 9.—Patient, 24 years old, 2 years and 10 months in service. There was defective hearing at the age of nine following a head injury. This condition has been stationary since. No hearing tension noted. Patient has organic disease.

VIII

PENICILLIN THERAPY IN EXTERNAL OTITIS

BEN H. SENTURIA, M.D.

ST. LOUIS, MO.

The widening scope of our military activities in tropical and subtropical areas has resulted in an increase in the number of cases of external otitis which must be treated by Service physicians. It may be further anticipated that increasing amounts of penicillin will be available for use in such minor diseases as external otitis.

Since penicillin has been effective in so many varied diseases, it is not surprising that numerous inquiries have been received concerning its possible use in the treatment of acute external otitis. Although conclusive experimental evaluation of any recommended therapy is difficult and time consuming, recently acquired data appears to delimit clearly the indications and contraindications for the use of penicillin in this disease entity.

Specific treatment of any disease usually waits on the discovery of the causative agent. There still exists, however, a sharp division of opinion among otologists as to the etiology of external otitis One school of thought believes that in hot humid countries this disease is caused by such saprophytic fungi as Aspergillus, Penicillium, Mucor, or Monilia. The other group considers external otitis to be a bacterial disease. Fungi which are cultured from some ears are thus believed to have been present before the onset of the infection or to have been implanted on "fertile soil" once the bacterial infection had caused maceration of the epithelium lining the external auditory canal and, with few exceptions, are of little importance in the etiology of external otitis.

When the literature was reviewed in an effort to establish one or more organisms as the cause of this disease, it was observed that reports from China,[1] India,[2] Malta,[3] Hawaii,[4] and southern United States[5-8] emphasized the finding of fungi in a large percentage of cases. Of equal importance, for purposes of this report, were the

---

From the AAF School of Aviation Medicine, Randolph Field, Texas, and the Department of Otolaryngology, Washington University School of Medicine and the Oscar Johnson Institute, Saint Louis, Missouri.

references to the high incidence of gram-negative bacilli which resembled the Pseudomonas group of organisms.[3, 9]

In a recent report on 135 cases of external otitis observed at Randolph Field, Texas,[9] and in reports from New Guinea[10] and the Solomon Islands,[11] it was shown that fungi were found in a small number of cases and that gram-negative bacilli (especially the Pseudomonas group) were found in a very large percentage of cases observed in these tropical and subtropical areas.

On the basis of our present knowledge of the action of penicillin,[12, 13] these bacteriologic and mycologic findings suggested, a priori, that penicillin would not be effective in those cases of external otitis in which the disease was caused by, or the predominating infecting organism was, a gram-negative bacillus or a saprophytic fungus.

In order to determine the accuracy of this hypothesis, a series of in vitro experiments were conducted, and a small number of clinical cases of external otitis was treated with penicillin solution and penicillin-sulfanilamide powder.

Subjects complaining of external otitis were selected at random from a large series of cases referred for diagnosis and treatment. Each was classified on the basis of history and physical findings[9] as one of the following types of infection:

Acute	(Circumscribed	
	(Diffuse	(Mild)
		(Moderate)
		(Severe)
Chronic	(Eczematoid	
	(Desquamative)*	

Routine cultures were taken directly from infected ear canals by means of sterile applicators and placed on Sabouraud's and blood agar media. All cultures were observed and reported in a manner previously outlined. Repeated post-therapy cultures were taken in an attempt to follow changes in the bacterial flora resulting from treatment.

Penicillin was diluted with sterile physiological solution of sodium chloride or with sulfanilamide powder, barium sulphate, or talc to give various dilutions of penicillin from 250 to 2000 Oxford units per gram or per cubic centimeter. Diluted penicillin was kept in a

*In previous reports, chronic desquamative external otitis has been referred to as "chronic suppurative external otitis."

refrigerator at all times except when in use and was discarded after a period of 30 days.

Local applications of penicillin to the ear canal were achieved by the use of the "drop" or "cotton pack" methods. When the drop method was used, the head was placed on a table with the infected ear up. One cc. of penicillin solution was instilled into the canal and allowed to remain there for five minutes. A cotton wick was then inserted and left in place until the next treatment. The cotton pack method consisted of inserting a cotton wick, saturated with penicillin solution, into the ear canal where it remained until the time of the next examination.

In vitro experiments were performed with pure cultures of bacteria and fungi obtained from cases of external otitis. The in vitro methods consisted of the agar cup plate technique and the quadrant streak method. The former is widely used to evaluate the relative merits of germicides.[12, 13] The latter was utilized in an effort to duplicate as closely as possible the methods used in the clinical treatment of external otitis.[14]

The agar cup plate technique employed pour plates which were prepared by inoculating approximately 20 cc. of melted nutrient agar which had been cooled to about 45° C. with a 2 mm. loopful of a 24-hour culture of the bacteria to be tested. To determine the inhibiting effect of penicillin solution on fungi found in cases of external otitis, pour plates of Sabouraud's dextrose agar were prepared in the same manner and inoculated with a 2 mm. loopful of the spores of the fungus.

The plates were allowed to solidify completely. A cup, one inch in diameter, was cut in the center of each plate by means of a test tube which was sterilized by heating to red heat in a free flame and allowed to cool. The portion of the medium inside the circle was removed with the aid of a sterile spatula. The bottom of the cup thus formed was then sealed with two drops of sterile melted agar added from a sterile pipette. Five drops of a solution of the chemotherapeutic agent to be tested were instilled into the cup from a pipette, and the plate was then incubated at 37° C. and observed for zones of inhibition after 24 and 72 hours.

The quadrant streak method was used to determine the in vitro effect of penicillin-sulfanilamide powder upon bacteria found in cases of external otitis. Blood agar plates (prepared from blood agar base to which 5% defibrinated human blood was added) were marked off into quadrants, and a 2 mm. loopful of a 24-hour culture of the

organism was streaked from one quadrant to the next without reflaming the loop.

Fungi were tested in the same manner on Sabouraud's dextrose agar, one loopful of the spores being implanted onto the center of each quadrant.

The mixture of penicillin-sulfanilamide powder containing different concentrations of penicillin (250 U/gm., 500 U/gm., etc.) was distributed over the inoculated plates by means of a powder shaker or insufflator. Control plates sprinkled with sulfanilamide powder alone and penicillin-barium sulphate mixture were set up in the same manner. All plates were incubated at 37° C. and observed for bacterial or fungus growth after 24 and 72 hours.

In Vitro Results. Using the agar cup plate technique, Mucor corymbifer, Aspergillus fumigatus, Aspergillus niger, and Monilia albicans were not inhibited by 5 drops of penicillin (500 U/cc.) applied three times a day at 4-hour intervals for 2 days. Control plates treated with 5 drops of 50% cresatin in olive oil containing 1% thymol (a preparation widely used in the treatment of otomycosis) showed complete inhibition of all fungi.

Pure cultures of three strains of Pseudomonas organisms* and one of hemolytic Staphylococcus aureus obtained from cases of acute external otitis were set up using the agar cup technique. Control plates seeded with Staphylococcus aureus showed excellent inhibition. Five drops of penicillin containing 250, 500, 1000, 2000, 5000, 10,000, 25,000, 50,000, and 100,000 U/cc. were instilled into the plates containing the Pseudomonas organisms. No inhibition was present except in the plate receiving the 100,000 U/cc. This is in agreement with the well-recognized action of penicillin.[12, 13]

A series of streaked plates was prepared using a culture of hemolytic Staphylococcus aureus obtained from one of the chronic cases. These were dusted with sulfanilamide powder alone, penicillin in talc (500 U/gm.), and talc alone. Complete inhibition occurred with penicillin-talc, but no inhibition occurred on the sulfanilamide, talc, and untreated control plates.

A number of other experiments were performed, using the streak method to test the action of powdered sulfanilamide, penicillin-barium sulphate, and penicillin-sulfanilamide against several strains

*Motile gram-negative bacilli, which showed characteristic green pigment and fruity odor, produced hemolysis on blood agar, proteolysis on blood serum media and fermented lactose.

of pyocyaneus-like organisms obtained from cases of acute external otitis. Sulfanilamide alone and the penicillin-sulfanilamide powder mixture (500 U/gm.) caused almost complete inhibition of growth, whereas penicillin-barium sulphate and the control plates showed full growth of three strains of Pseudomonas organisms tested. On the other hand, little inhibition occurred when penicillin-sulfanilamide powder mixtures were applied to other strains of Pseudomonas organisms obtained from acutely infected ear canals.

Clinical Results. Penicillin solution and penicillin-sulfonamide powder mixtures were used in the treatment of 32 cases (ears) of external otitis which showed various types and grades of infection. Cases were selected, typed, and graded on the basis of clinical findings. Those cases showing severe signs and symptoms were usually started immediately on penicillin solution intramuscularly; mild and moderate cases received penicillin drops or the powder mixture.

Penicillin solution was applied topically to 11 ears (Cases No. 1 to No. 11, Table I) in the form of drops or by means of a cotton wick saturated with penicillin. Unsatisfactory therapeutic results were obtained in 10 out of the 11 ears treated with penicillin solutions (500 to 2000 Oxford units per cc.). Two ears (Cases No. 3 and No. 6) demonstrated improvement during the first four to six hours and then became much worse during treatment, resulting in a change of diagnosis from mild diffuse to severe diffuse external otitis. Cases No. 4, No. 9, and No. 11 developed vesicular dermatitis in and about the auricle which appeared to be directly attributable to the use of penicillin. (See Appendix).

One ear (Case No. 7) showing Staphylococci and Pseudomonas organisms improved under penicillin therapy. This case which had been diagnosed as acute severe diffuse external otitis had been treated previously with a 4:2 mixture of sulfanilamide and zinc peroxide[16] without subsidence of pain and obtained almost immediate relief when penicillin therapy was instituted.

Nine subjects (Cases No. 12 to No. 20, Table II) were treated with intramuscular injections of penicillin (20,000 units every 3 hours). In each case, where the findings permitted a diagnosis of circumscribed external otitis (Cases No. 12 to No. 17), rapid subsidence of pain and cellulitis occurred. Well-localized fluctuant abscesses developed in each of these ears within 24 to 48 hours. Spontaneous drainage occurred in most of the cases although incision and drainage of the abscess was occasionally required. Staphylococci were obtained in pure culture or were the predominant organisms on culture in all of these cases of circumscribed external otitis.

Two ears (Cases No. 18 and No. 19) diagnosed as acute diffuse external otitis and showing pure cultures of Pseudomonas organisms were given intensive intramuscular penicillin therapy for three to four days. These patients showed a persistence of pain, edema of the canal and profuse serous drainage. Improvement occurred only after local therapy was instituted. One subject (Case No. 20) showed a mixed infection and responded slowly to combined therapy after intensive treatment.

Seven patients (Cases No. 26 to No. 32, Table IV) were treated with the penicillin-sulfanilamide powder (500 U/gm.) by means of the insufflation technique.[18] Good results were obtained in the three mild cases, but poor therapeutic results were demonstrated in the one moderate diffuse ear infection treated with this powder. In the chronic eczematoid cases from which hemolytic Staphylococcus aureus was cultured, no relief of symptoms was observed.

DISCUSSION

It has been demonstrated by numerous investigators that penicillin is active against many of the gram-positive organisms, but very few of the gram-negative bacilli. Consequently, if penicillin therapy in external otitis is contemplated, cultures of infected ear canals should first be obtained to determine the nature of the bacterial flora. If the smears and culture reports, which are usually available within 24 to 48 hours, indicate that organisms which are probably penicillin sensitive are present, then penicillin therapy may be of value. If gram-negative bacilli are present in large numbers in the cultures obtained from the infected ear canal, poor therapeutic results may be anticipated. Gram-negative bacilli present in the ear canal may or may not be the etiological agent, yet the use of this antibiotic will be vitiated, if they are of such a nature as to destroy penicillin.[19, 20]

A very large percentage of the cases in this series showed gram-negative rods among the organisms obtained on cultures prior to treatment with penicillin. If the acute circumscribed cases are omitted, these findings are in agreement with the observations in a larger series of cases reported recently.[9]

Cognizance is taken of recent work[17] which demonstrated that many organisms, such as the gram-negative intestinal group, which are considered penicillin resistant, will be inhibited by high concentrations of penicillin (5-20 U/cc.). Blood levels of this magnitude cannot be obtained easily. However, if high concentrations of penicillin are maintained locally in direct contact with the infecting or-

ganisms in the ear canal, good results may be obtained in a small number of cases despite the presence of relatively resistant organisms. This probably explains the good results obtained in our cases showing infection with gram-negative rods.

Recurrent circumscribed lesions of the external auditory canal are often seen and may be expected to respond promptly to the use of local penicillin therapy since Staphylococci, which are frequently sensitive to penicillin, are cultured from these lesions. If the lesion is treated early, abscess formation may possibly be prevented. If seen later in the course of the disease, rapid localization of the original lesion may occur, and involvement of other hair follicles in the ear canal may be avoided by the use of penicillin solution. It is noteworthy, however, that in this group of cases in no instance did the use of topical penicillin prevent abscess formation, and in two cases this form of therapy failed to produce improvement.

In direct contrast to these equivocal results, the response to intramuscular penicillin in those cases of circumscribed external otitis from which Staphylococci were cultured was dramatic. Rapid relief of pain and localization of abscess occurred in all cases. In several cases where no improvement was obtained from penicillin applied topically, rapid response occurred following the use of intramuscular penicillin.

Penicillin-sulfanilamide powder mixtures have been used extensively for local application[21, 22] and may be of distinct value in mild external otitis. However, in the case of some virulent strains of Pseudomonas organisms, neither penicillin nor sulfanilamide alone, or in combination, will effectively inhibit the growth of these organisms.

Penicillin in solution, or in combination with mild ointments, such as the carbowaxes, appears as a potent therapeutic weapon for use in the chronic eczematoid cases which are infected primarily or secondarily with penicillin-sensitive Staphylococci. It is doubtful, however, if many permanent cures will be obtained with this type of external otitis until the underlying mechanism of the condition is more fully understood.

Several reports are now available in the literature describing toxic reactions[21, 23, 24] and sensitivity to penicillin or some impurity contained in the present product.[23, 24] In the appendix is included a complete report of Cases No. 9 and No. 11 (ears), which are considered proven examples of hypersensitivity to penicillin.

SUMMARY

1. Penicillin solutions and penicillin sulfonamide mixtures were used in the treatment of 32 cases (ears) of external otitis.

2. Penicillin solution applied topically produced unsatisfactory therapeutic results in 10 out of 11 cases diagnosed as acute diffuse, chronic eczematoid, or chronic desquamative external otitis.

3. Rapid subsidence of pain and cellulitis and formation of well-localized abscesses occurred within 24 to 48 hours in six cases of circumscribed external otitis treated with intramuscular penicillin.

4. Seven patients were treated with penicillin-sulfanilamide mixtures by means of the insufflation technique. Equivocal results were obtained.

5. If smear and cultures indicate that penicillin-sensitive organisms predominate in the external auditory canal, good therapeutic results may be anticipated from the use of intramuscular penicillin and possibly from the topical application of this antibiotic.

6. A large percentage of the cases in this series showed on culture a predominance of gram-negative rods. In vitro and clinical results confirmed the prediction that penicillin would not be effective in those cases of external otitis in which the disease was caused by, or the predominating organism was, a gram-negative bacillus.

Acknowledgment is made to Major Stanley F. Hampton, M.C. Chief of Allergy Section, AAF Regional Hospital, San Antonio Aviation Cadet Center, for assistance with the allergic work-up, to Corporal Fred Wolf for identification of fungi, to Mrs. P. Anderson, Mrs. R. Blake, Miss Reta Gieskin, Miss Maureen Garland, and Sergeant Stanley Hubert for technical assistance.

WASHINGTON UNIVERSITY
SCHOOL OF MEDICINE.

APPENDIX I

A CASE OF PENICILLIN HYPERSENSITIVITY—CASES (EARS) 9 AND 11

PATIENT: W.B.F. AGE: 25.

ADMITTED: Station Hospital, Randolph Field, Texas, 22 March 1944.

CHIEF COMPLAINT: Intermittent drainage from left ear canal.

FAMILY HISTORY: Mother has had urticaria; otherwise negative.

PREVIOUS PERSONAL HISTORY: Scarlet fever at the age of 17 years. No history of drug sensitivity or of allergic manifestations. Fracture of second metatarsal bone in 1943 and of fifth finger of right hand in 1937. Denies history of venereal disease. Had used sulfonamide nose drops about two years ago with good results. Only other sulfonamide medication was a test dose

of four tablets (grains 30) for respiratory prophylaxis in December 1943 without ill effects. No previous exposure to penicillin.

PRESENT HISTORY: Left ear started draining pus about December 1943 unassociated with pain; drainage was intermittent. Treatment before hospitalization at the Station Hospital, Randolph Field, Texas, consisted of sulfanilamide powder and merthiolate. There was no previous ear trouble except accumulation of wax in the canal. Toothpicks and matches were used to remove excess wax. No swimming for past two months.

PHYSICAL EXAMINATION ON ADMISSION: All findings negative except:

Head: Asymmetry of face with fullness of left malar region and broadness of the root of the nose.

Nose: Mild nasal congestion.

Throat: Tonsils mildly hypertrophic.

Ears: Left auditory canal contains purulent discharge. Tympanic membrane is reddened with loss of all detail. Concha excoriated. (Right ear canal became similarly infected during period of hospitalization.)

LABORATORY DATA:

Blood: White blood count, 5,950. Differential: neutrophiles 54, lymphocytes 36, monocytes 6, eosinophiles 4.

Urine: Negative.

Culture: Pus from ears: Pseudomonas organisms, Staphylococcus aureus. Fungi— no growth on Sabouraud's media.

Glucose Tolerance Tests: Mildly diminished carbohydrate tolerance, but repeated tests after high carbohydrate diet for one week showed negative Exton-Rose and standard tests.

X-rays: Chest—negative. Mastoids—slight periantral haziness bilaterally, but otherwise well aerated. Sinuses—paranasal sinuses are normal throughout except for slight mucosal thickening in the right maxillary sinus.

DIAGNOSIS: Otitis externa, acute, diffuse, bilateral.

TREATMENT: *Left ear* (1) Penicillin diluted with pyrogen-free physiologic sodium chloride solution and applied on cotton wick. One cc. (500 U/cc.) every 3 hours for 4 treatments. Profuse discharge and excoriation of concha noted 36 hours after beginning of treatment. (2) Zephiran chloride (1:1000), and boric and alcohol (70%) drops finally resulted in cure. *Right ear* (1) Penicillin diluted with pyrogen-free physiologic sodium chloride solution and applied on cotton wick. One cc. (2000 U/cc.) instilled every 2 hours for 2 days during waking hours, resulting in profuse discharge, redness, edema of the canal and vesiculation of the concha and the lobule. (2) Zephiran chloride, alcohol, Castellani's solution, cresatin, and sulfonamide mixture failed to control the infection during the course of the disease.

SUMMARY OF ALLERGIC EXAMINATION:

Bacteriology: Culture of both ears revealed heavy growth of Staphylococcus aureus and of Pseudomonas organisms.

Intradermal Skin Tests: Negative to freshly prepared solution of penicillin (10,000 Oxford U/cc.). Negative tests to common pollens, inhalants, and foods. Vaccines of the above named organisms containing approximately 1,500,000,000 per cc. were tested intradermally. The pyocyaneous vaccine (1-100 dilution) revealed a 2+ to 3+ reaction with edema approximately 3 cm. in diameter 24 hours after testing. Skin tests with

the Staphylococcus aureus vaccine gave a similar reaction with a 1-10 dilution and a less intensive reaction with a 1-100 dilution.

Patch Test: A 2+ reaction in 24 hours was obtained with a freshly dissolved solution of penicillin containing 10,000 Oxford U/cc. This was repeated with penicillin solution prepared 10 days previously. Positive patch tests were obtained to both the concentrated solution (10,000 Oxford U/cc.) and a 1-10 dilution (1000 U/cc.) while tests with a 1-100 dilution (100 U/cc.) were negative. Patch test with a Wyeth preparation (2000 U/cc.) caused local itching, redness, and edema confined strictly to the area of contact. The patient and two normal subjects were subsequently tested in a similar manner with the same concentration of Squibb penicillin. After 48 hours the patient developed itching, a patchy redness, and folliculitis in the area of penicillin contact. No reactions occurred in the normal subjects.

Passive Transfer Tests: Prausnitz-Kustner tests were performed with the patient's blood serum and were entirely negative to freshly dissolved penicillin solution (10,000 U/cc.). Penicillin mold and patch tests applied in the passive transfer technique with freshly dissolved penicillin solution (10,000 Oxford U.) were also negative.

TABLE I

CASES OF EXTERNAL OTITIS TREATED LOCALLY WITH PENICILLIN SOLUTIONS

Case No.	Grade of Infection	Infecting Organisms	Solution	Method	Reactions
1.	Acute, mild, diffuse	Pseudomonas (sp.)	2000 U/cc.	Drops. 3 applications at 3 hr. intervals.	Ear canal blocked with profuse discharge shortly after treatment.
2.	Acute, moderate, diffuse	Pseudomonas (sp.) Gram-negative rod	2000 U/cc.	Pack. 3 applications at 3 hr. intervals.	Far worse after treatment.
3.	Acute, moderate, diffuse	Pseudomonas (sp.)	2000 U/cc.	Drops and pack at 2 hr. intervals for 3 days.	Temporary improvement and recurrence of pain. Persistent edema of ear canal.
4.	Acute, moderate, diffuse	Pseudomonas (sp.) Gram-positive rod	2000 U/cc. 1000 U/cc.	Drops at 2 hr. intervals for 9 days.	Slow subsidence with persistence of green secretion over period of 9 days. Papular rash.
5.	Acute, moderate, diffuse	Pseudomonas (sp.) Micrococcus tetragenus Sarcina	2000 U/cc.	Drops. 4 applications at 2 hr. intervals.	Poor results.
6.	Acute, severe, diffuse	Pseudomonas (sp.)	2000 U/cc.	Drops 4 times daily for 2 days.	Temporary improvement, then worse.
7.	Acute, severe, diffuse	Pseudomonas (sp.) Staphylococcus	2000 U/cc.	Pack applied for period of 24 hrs.	Relief within several hours. Had been treated with 4:2.

TABLE I—(*Continued*)

Case No.	Grade of Infection	Infecting Organisms	Solution	Method	Reactions
8.	Chronic, eczematoid	Staphylococcus aureus	2000 U/cc.	Drops 4 times daily for 4 days.	Failed to relieve symptoms.
9.	Chronic, desquamative	Pseudomonas (sp.)	2000 U/cc. 500 U/cc.	Pack at 3 hr. intervals for 3 days.	Vesicular rash. Profuse green pus persisted.
10.	Chronic, eczematoid	Staphylococcus aureus Gram-negative rod	2000 U/cc.	Drops 4 times daily for 4 days.	Poor results.
11.	Chronic, desquamative	Pseudomonas (sp.)	2000 U/cc.	Pack at 2 hr. intervals for 2 days.	Developed vesicular dermatitis which spread to face. Profuse green pus present for 2 days after penicillin stopped.

TABLE II

CASES OF EXTERNAL OTITIS TREATED WITH INTRAMUSCULAR PENICILLIN

Case No.	Type & Grade of Infection	Infecting Organisms	Treatment	Remarks
12.	Acute, circumscribed, severe	Staphylococcus aureus, hemolytic	20,000 U q. 3 hr. for 7 days.	Spontaneous drainage of one furuncle and incision of two others. Remarkable subsidence of pain and cellulitis following institution of penicillin therapy.
13.	Acute, circumscribed, moderate	Staphylococcus aureus, non-hemolytic	20,000 U q. 3 hr. for 4 days. 250 and 1000 U q. hr. locally for 6 days.	Poor response to local applications of penicillin. Almost immediate response to I.M. use; profuse spontaneous drainage.
14.	Acute, circumscribed, severe	Staphylococcus aureus, hemolytic	20,000 U q. 3 hr. for 3 days.	Good resolution of cellulitis; spontaneous drainage.
15.	Acute, circumscribed, moderate	Staphylococcus aureus, hemolytic	20,000 U q. 3 hr. for 3 days after local therapy unsuccessful.	No relief from penicillin or tyrothricin locally. Rapid response from I. M. penicillin.
16.	Acute, circumscribed, severe	Staphylococcus aureus, non-hemolytic; Staphylococcus albus, non-hemolytic	20,000 U q. 4 hr. for 16 injections, then pack (1000 U/cc.) daily for 3 days.	Progressive improvement; relief of pain in first 24 hours; spontaneous drainage.
17.	Acute, circumscribed, severe	Staphylococcus aureus, non-hemolytic	20,000 U q. 4 hr. for 3 days.	Remarkable subsidence of pain and cellulitis.

TABLE II--(*Continued*)

Case No.	Type & Grade of Infection	Infecting Organisms	Treatment	Remarks
18.	Acute, diffuse, severe	Pseudomonas (sp.)	20,000 U q. 3 hr. for 4 days.	Poor results under penicillin therapy; persistence of pain, edema and discharge until other therapy started.
19.	Acute, diffuse, severe	Pseudomonas (sp.)	20,000 U q. 3 hr. for 3 days.	Slight gradual improvement in edema; continued pain and discharge; other therapy instituted.
20.	Chronic eczematoid	Gram-negative rod Gram-positive bacillus	20,000 U q. 3 hr. for 3 days; 2000 U locally; boric ointment and ultraviolet therapy.	Good results after prolonged treatment.

TABLE III

CASES OF CIRCUMSCRIBED EXTERNAL OTITIS TREATED LOCALLY WITH PENICILLIN

Case No.	Type & Grade of Infection	Infecting Organisms	Treatment	Remarks
21.	Acute, circumscribed, mild	Staphylococcus aureus, hemolytic; Staphylococcus albus; Corynebacteria.	2000 U/cc. at 6 hr. intervals—3 applications.	Good results in eliminating pain and cellulitis; spontaneous drainage.
22.	Acute, circumscribed mild	Staphylococcus aureus, nonhemolytic, Corynebacteria.	1000 U/cc. at 24 hr. intervals—3 applications.	Rapid subsidence of cellulitis and pain; incision and drainage.
23.	Acute, circumscribed, mild	Staphylococcus aureus, hemolytic and non-hemolytic; Streptococcus, nonhemolytic; Corynebacteria.	1000 U/cc. b.i.d. for 4 days.	Rapid localization and relief of pain; spontaneous drainage.
24.	Acute, circumscribed, mild	Staphylococcus aureus, hemolytic.	1000 U/cc. once a day for 2 days.	No relief of cellulitis or pain.
25.	Acute, circumscribed, mild	Staphylococcus aureus, nonhemolytic.	1000 U/cc. once a day for 2 days.	Increasing pain until spontaneous draining after second day.

TABLE IV

CASES OF EXTERNAL OTITIS TREATED WITH PENICILLIN SULFANILAMIDE POWDER
(500 U/gm.)

Case No.	Grade of Infection	Infecting Organisms	No. Insufflations	Reactions
26.	Acute, mild, diffuse	Sporeformers, Staphylococcus albus non-hemolytic, Streptococcus non-hemolytic	One	Good results.
27.	Acute, mild, diffuse	Pseudomonas (sp.) Staphylococcus albus, Sarcina	One	Good results. Pseudomonas and Sarcina disappeared from cultures.
28.	Acute, mild, diffuse	Staphylococcus aureus	One	Rapid subsidence.
29.	Acute, moderate, diffuse	Pseudomonas (sp.)	One	Developed into severe desquamative infection.
30.	Chronic eczematoid	Staphylococcus aureus, hemolytic	Two	Initial improvement, but recurrence of symptoms.
31.	Chronic eczematoid	Gram-negative rod, Penicillium (sp.)	One	Ear improved but slightly painful for 48 hours.
32.	Chronic eczematoid	Staphylococcus aureus	One	Continued itching and dryness.

REFERENCES

1. Dunlap, A. M.: Otomycosis, Chinese M. J. 52:446, 1937.

2. Hatch, W. K., and Row, B.: Fungus Disease of the Ear, Lancet 2:1561, 1900.

3. Daggett, W. I.: Desquamative Otitis Externa in Malta, J. Laryng. & Otol. 57:427 (Oct.) 1942.

4. Trexler, Clarence W.: Otomycosis in Hawaii, Laryng. 45:106 (Feb.) 1935.

5. Gill, W. D.: Mycotic Infections in Otolaryngology, South. M. J. 31:678 (June) 1938.

6. Gill, King: Otitis External Mycotica, Arch. Otolaryng. 16:76 (July) 1932.

7. Dobes, W. L.: Moniliasis of the External Ear Canal, South. M. J. 36:614 (Sept.) 1943.

8. Simms, R. F.: Aspergillus Infection as the Cause of External Ear Diseases, South. M. J. 30:1224 (Dec.) 1937.

9. Senturia, B. H.: Etiology of External Otitis, Laryng. 55:277-293, 1945.

10. Quayle, A. F.: Otitis Externa in New Guinea, Med. J. of Australia 2:228 (Sept. 2) 1944.

11. Beach, E. W., and Hamilton, L. L.: Tropical Otitis Externa; Ear Fungus, U. S. Naval Med. Bull. 44:599-602 (Mar.) 1945.

12. Fleming, Alexander: On the Antibacterial Action of Cultures of a Penicillium, with Special Reference to Their Use in the Isolation of B. Influenzae, Brit. J. Exper. Path. 10:226, 1929.

13. Dawson, M. H., Hobby, G. L., Meyer, Karl, and Chaffee, Eleanor: Penicillin as a Chemotherapeutic Agent, Ann. Int. Med. 19:707, 1943.

14. Allen, A. Wm., and Wright, I. S.: Determination of the Value of Antiseptics, J. A. M. A. 96:920 (Mar.) 1931.

15. Ruehle, G. L. A., and Brewer, C. M.: U. S. Food and Drug Administration Methods of Testing Antiseptics and Disinfectants, Cir. No. 198 (Dec.) 1931.

16. Senturia, B. H., and Wolf, F. T.: The Action of Sulfonamides on Fungi Isolated from Cases of Otomycosis, Report No. 1, Project No. 212 (Jan.) 1944, AAF School of Aviation Medicine, Randolph Field, Texas.

17. Thomas, A. R., and Levine, Max: Some Effects of Penicillin on Intestinal Bacteria, to be published.

18. Senturia, B. H.: Treatment of External Otitis I.—Local Sulfonamide Therapy, Laryng. (June) 1944.

19. Harper, G. J.: Inhibition of Penicillin in Routine Culture Media, Lancet 2:569-571 (Nov. 6) 1943.

20. Abraham, E. P., and Chain, E.: An Enzyme from Bacteria Able to Destroy Penicillin, Nature 146:837 (Dec. 28) 1940 (letter to editor).

21. Keefer, C. S., Blake, F. G., Marshal, E. K., Jr., Lockwood, J. S., and Wood, W. B., Jr.: Penicillin in the Treatment of Infections, J. A. M. A. 122:1217 (Aug.) 1943.

22. Florey, H. W., and Florey, M. E.: General and Local Administration of Penicillin, Lancet 1:387, 1943.

23. Dawson, M. H., and Hobby, G. L.: The Clinical Use of Penicillin, J. A. M. A. 124:611 (Mar.) 1944.

24. Lyons, Champ: Penicillin Therapy of Surgical Infections in the U. S. Army, J. A. M. A. 123:1007 (Dec.) 1943.

25. McCloskey, W. T., and Smith, M. I.: Experiments on the Sensitizing Properties of Penicillin, Proc. Soc. Exper. Biol. and Med., Vol. 57, Nov., 1944.

26. Welch, Henry, and Rostenberg, Adolph, Jr.: Hypersensitivity of the Tuberculin Type to Crystalline Penicillin Sodium, J. A. M. A. 126:10 (Sept.) 1944.

IX

OTOLARYNGOLOGY IN THE EUROPEAN THEATER OF WORLD WAR II

COLONEL NORTON CANFIELD

MEDICAL CORPS, ARMY OF THE UNITED STATES

As an integral part of a complete medical service for the sick and wounded soldier, highly skilled methods and techniques are recognized as essential. The Army Medical Corps during this war has recognized and encouraged medical specialists and, within the limitations of Army regulations, has made them available for the care of special cases. During peacetime the civilian specialist has had little opportunity to observe, influence or profit by Army specialty practice. The Regular Army specialist has had some opportunity to progress, but in many instances where unusual cases appear civilian specialists are consulted and often provide the necessary care. Military advancement in rank of a Regular Army specialist, if he keeps his assignment as a medical professional officer, is possible only to a certain degree, after which if a higher rank has been earned by the officer, he is frequently no longer a practicing specialist. He is then assigned to some administrative post and officers of lower rank carry on. The result is that there are then only a few high ranking specialist practitioners in the Regular Army. This tends, first, to discourage certain special ability, and, second, it deprives the Army of having widely experienced officers among some groups of practicing specialists.

When war comes, the expansion due to the emergency requires that Regular officers be advanced in rank to hold administrative posts, thereby removing them from their professional duties. This in turn requires that the civilian doctor supply the specialists to fill these posts. Hence the civilian specialty group comes into existence during war, made up almost entirely of newly recruited Army officers. To date no provision has been made to keep a Regular Army officer in high rank on purely professional duties in the specialty of otolaryngology, and no civilian consultant in this field was on duty at The Surgeon General's Office until July 1944. However, civilian consultants had been appointed but had no influence on professional policy. These men were not well acquainted with Army medical

organization and were of little value in the administration of the specialty, professionally or otherwise.

By a decision of the Chief Surgeon of the European Theater of Operations, a Professional Service Division was maintained in his office from July 1942 to August 1945. This consisted of an administrative chief of section with the rank of Colonel, a senior consultant in surgery and in medicine both with the rank of Colonel, two other senior consultants on the medical side in psychiatry and dermatology, and in surgery a senior consultant in general surgery, orthopedics, neurosurgery, plastic surgery, oromaxillary surgery and also in otolaryngology, ophthalmology, anesthesiology, and radiology, with the preliminary rank of Lt. Colonel, being later promoted to Colonel. This group carried on its duties by intimate contact with the medical officers in the Theater who were actually treating patients. It advised the Chief Surgeon on matters of policy concerning medical and surgical care in all its phases including equipment, personnel, hospital management and medical supplies. Frequent professional directives were written by members of this Division, and as the war progressed changes of professional policy advocated by this Division were implemented by the Chief Surgeon. Although chiefly professional in character, the Division touched on every phase of medical care which affected the recovery of the sick and wounded soldier. Under the able leadership of Brig. General Elliot Cutler in surgery and Colonel William Middleton in medicine, its work was constantly encouraged by the Chief Surgeon and hampered only by the exigencies of the war and the administrative policies of higher echelons of command.

In the European Theater, not counting those administrative officers of the Regular Corps, who left specialty practice as their rank increased, and officers of the Air Force Medical Corps previously trained in a specialty, approximately 95 per cent of the otolaryngology specialists were assigned so that their principal duty was the practice of their specialty in hospitals and dispensaries. This did not mean that their specialty ability was actually used in the most efficient manner; this is so because of the nature of Army medicine which must be practiced as it now is as long as the present organization of military hospitals is maintained. Considering the nonprofessional duties, the time used in moving from place to place, the many patients seen which could be adequately attended by less well-trained officers, an estimation of approximately 50 per cent efficiency for this group is correct. Methods of considerably increasing this efficiency would constitute a definite advance in military medicine. The specialist, however, in this war for the first time has had excellent recognition

compared to previous wars, and it remains only for the civilian and the military medical authorities to continue the close cooperation in peacetime which has been so effective during the war.

During the European campaign 364 medical officers whose previous training in a specialty had been sufficient to qualify them for specialty rating in otolaryngology were sent by The Surgeon General's Office. Of these 118 had been certified by the American Board of Otolaryngology, 143 had a rating by The Surgeon General's Office in both otolaryngology and ophthalmology, of which 17 were certified by the Board of Otolaryngology and 3 by both the Boards of Ophthalmology and Otolaryngology.

The policy of The Surgeon General to rate each specialist according to his ability divided the men into four groups.

Group A consisted of officers with civilian or military background of recognized and outstanding ability in a specialty, for example, officers who were professors or heads of the department in large teaching centers. There were six of these.

Group B were officers with superior training and demonstrated ability. Classification in this group indicates a probable training period of one year as an intern and a three-year residency or fellowship, devoted to the specialty in a recognized teaching center. Of these there were 54.

Group C included two types of officers, first, those who had recently completed periods of training, including one year as an intern and two to three years as a resident in a specialty and, second, officers with shorter periods of training, but with backgrounds of mature experience. There were 186 so graded.

In Group D were officers who had recently completed periods of training, including one year as an intern and one year as a resident, or officers who had demonstrated some ability in a specialty, or officers with shorter periods of training but with a minor proportion of practice devoted to a specialty by giving it particular attention. There were 119 of these.

The otolaryngologic specialty officer requirements according to existing regulations for the number of hospitals sent to the European Theater were of Class A, 1, Class B, 0, Class C, 401, and Class D, 63. The regulations up until July 3, 1944, stated that either the otolaryngologist or the opthalmologist in a general hospital could attain a rank as high as Major. On this date the regulation was published calling

for the otolaryngologists in all overseas hospitals up to the 2000-bed general hospital to have the grade of Captain, the ophthalmologists to have the grade of Major. Some commanding officers, however, did recommend promotions to the rank of Major for the otolaryngologists in a general hospital. Approximately six were so promoted. No otolaryngologists assigned to a hospital was promoted beyond the rank of Major on his professional ability. Two otolaryngologists were promoted to the rank of Lieutenant Colonel on the basis of their duties as executive officers. This situation was the source of considerable concern and no little anxiety on the part of the Theater Surgeon and the individual specialists, when it was also directed that the chiefs of the laboratory, neuropsychiatry, dentistry, radiology and nursing services all had the possibility of promotion to Lieutenant Colonel in 1000-, 1500- or 2000-bed hospitals. A comparable rank for comparable previous training, therefore, was extremely difficult for the otolaryngologists.

The organization of all hospitals directed that otolaryngology and ophthalmology should constitute a separate section of general surgery, with either the ophthalmologist or otolaryngologist being the chief of the section. This in many instances was satisfactory, but in some hospitals the chief of surgery was not sympathetic with, or cognizant of, the problems of the specialty. Hence, in these hospitals this special service was not up to standard. Although approximately ten patients were treated by otolaryngologists "medically" for one on whom an operation was performed the chief of surgery had authority to designate which patients were to be subjected to operation. This resulted in at least six general hospitals having a policy of making the section of ophthalmology and otolaryngology separate from the surgical service and directly responsible to the commanding officer. This occurred usually when the specialist was a more mature and experienced officer than the chief of surgery.

Out-patient service in all station and general hospitals located near noncombat troops was extensive and a large proportion of the time of the specialist was used to handle this load. In the largest of these clinics 1,800 patients per month for a period of 22 months went through the otolaryngologic clinic. This load was only exceeded in a few hospitals by the orthopedic out-patient service.

Training of specialists in the European Theater was not permitted by a policy of the chief surgeon. This was so ordered because it was believed that hospitals to be sent to the theater would be adequately supplied with satisfactory specialty officers. However, during the last 14 months before VE-Day, 30 general hospitals were sent

without any officers specially training in otolaryngology. This resulted in a distribution problem which was never adequately solved for three reasons. First, the number of officers available did not equal the number of assignments where specialists were required, second, attrition of officers was not compensated for by replacement, and third, because of considerations of military rank, hospital organization, and higher echelon regulations movement of offiecrs from one unit to another was inordinately slow. To overcome these deficiencies, however, a system of regional consultants was instituted.

With the concentration of hospitals that were operating in England, it was obvious that if each unit could not be self-sufficient, a system of cooperation for the care of special cases would accomplish the same purpose. Hospital centers were established and patients sent from one hospital to another if specialty care was indicated. In each center a regional consultant was appointed and was responsible for knowledge and recommendations concerning the specialty care of all patients within his group. This made it possible for some hospitals to function properly without their full complement of specialty personnel. Seven such centers with from 12 to 22 hospitals were in operation in England shortly before D-Day and until the theater was rapidly contracted. The same policy was established on the continent as the military situation permitted, but because of organizational difficulties inherent in an advancing army, the program was never as well established as in England. Too much praise and commendation cannot be attributed to these regional consultants. They served, in addition to their other duties, as chiefs of sections in general hospitals and at times with lack of adequate transportation and sympathetic understanding on the part of the hospital centers and unit commanders, they sent in reports, advised changes of personnel and themselves actually assumed responsibility for difficult cases. So effective was this system that expansion of it and a training program in connection with it could have adequately solved all of the personnel deficiencies inherent in a campaign similar to that in the European Theater.

Association with British Specialists. Little was it realized, until the hospitals landed in England, that such a warm welcome was waiting from our British colleagues. Many instances of generosity and friendliness during the first few months will gradually be related as stories of the individual men are told. Wherever hospitals were near those of the British we had but to make known our needs and they were fulfilled. In the early days the specialty instrument supply suffered from sinkings in the Atlantic. Hospitals were dependent

upon the local supply, which seldom failed. Officially through the senior consultant in otolaryngology of the Royal Army Medical Corps, Brigadier Myles Formby, many items were made available and at one time it was contemplated relying entirely on British supply for most items of special apparatus. The submarine was a menace which was difficult to predict, but fortunately it did not curtail essential medical operations except for a short time.

Not only was apparatus forthcoming but professional help and guidance was generously offered and enthusiastically received. All British medical meetings were open to Americans, licensure was permitted, and specialty assemblies in large cities were attended by many of our officers. Long will be the memories of the meetings of the Royal Society in London where six times a year the Sections of Otology and Laryngology welcomed the American specialists. On such occasions the discussions were often lively. Some Americans presented papers. The pleasant memory of such colleagues as Negus, Colledge, Cawthorne, Dickson, Bateman, Formby, Fry, Snag, Mollison, Hallpike, Davis, Martin, and Simpson-Hall will live for years as the high spots of our professional activities in England and Northern Ireland. Even after we moved across the Channel men returned to the meetings in London. A fine banquet which climaxed our British association was attended by about 40 American officers in June of 1945.

On the continent Paris offered facilities other than those of diversion and the monthly meetings of the Societé Otorhinolaryngolagie des Hôpitau de Paris was warm in its welcome. LeMaitre, Ramadier, Moulonguet, Aubin and others tolerated the lack of ability with the French language and the meetings were attended by those who could be relieved of their official duties. The University of Liege offered its clinic to those in that area, and many other individual contacts were made by officers in neighboring hospitals, medical schools and allied medical installations.

Equipment. Individual items of equipment were for the most part excellent, but the selection of some instruments was obviously ill-advised. Improvisation, which is indigenous in Americans, filled in many of the deficiencies and at the end there were only about one dozen items which constituted the list in the final recommendation to The Surgeon General's Office.

Clinical Phases. Before the fighting started in Normandy, specialty service was much the same as in any community except that accidents were somewhat more frequent. Almost no children were

treated and the diseases of the age group above 40 were rare. A few exceedingly interesting cases were encountered, such as one of Hodgkin's disease of the larynx involving the vocal cord, four cases of carcinoma of the larynx, a few severe exacerbations of chronic sinusitis, a case of osteomyelitis of the cervical vertebra and several cases of osteomyelitis of the skull, one of which cleared completely with penicillin without extensive operation although there was a large extradural abscess.

The use of sulfonamides for the control of acute otitis media and sinusitis has produced varying reports from different specialists. From the total experience it is fair to say that for controlling the complicating spread of infection to neighboring tissues the drug was of extreme value. For the treatment of acute otitis media after suppuration had occurred it was of questionable value. The most severe complications from mastoid infections were in patients nearly all of whom had had some sulfonamides in the course of treatment, and the masking effect, giving false security in an individual case, was frequent. This came to our attention on many occasions, especially when a patient was treated in the forward areas and residual infection was slight, but definitely present. There is no doubt but that many chronic suppurating ears now exist because they became nearly asymptomatic under the care of non-specialists, but in reality had smouldering infections which did not completely heal or which did or will become serious later. The damage to hearing is seldom evaluated in these cases but it will constitute a considerable proportion of the total damage to this organ.

Important also was the false sense of security obtained in the forward areas by the use of both sulfonamides and penicillin in wounds of the nose and sinuses. In many cases a failure to thoroughly clean the sinuses of debris of all kinds did not interfere with the healing of the skin wounds over the sinuses, but led later to suppurating infection and distortion which interfered with the normal emptying of the sinus at some later date.

These cases often could not be treated by sinus specialists and with evacuation policies which were necessary to clear the beds for the more seriously injured, many patients were sent to the rear, appearing to be doing well but in reality harboring potential sources of later complications. So frequent were these cases that instructions from the Chief Surgeon's office included urgent advice to be certain of the inside of any paranasal sinus whose radiograph showed increased density, in spite of the outward appearance of the patient. These cases will continually appear and because this war resulted in

so many non-radiopaque foreign bodies acting as secondary missiles, the same advice will apply to patients long after they are discharged from the Army.

After patients began coming back in large numbers from the continent, otolaryngologists were pressed into service for other emergency service. In transient hospitals where only nontransportable cases were held, not much otolaryngology was done. These patients were routed to hospitals further to the rear and where the system of hospitals centers was in operation, certain cases were collected in designated hospitals for their definitive care.

The many man-hours lost in the Army from Vincent's infection of the tonsil led Major Gordon Hoople to evolve a rapid treatment for these cases. Extreme care in cleansing the ulcer by suction and scrubbing, followed by application of sodium perborate powder, reduced the average stay in the hospital in these cases from an average of seven to less than two days. This officer also described a method of reducing zygomatic fractures by intranasal pressure with a sound passed through the inferior meatus, thereby avoiding an external or intra-oral approach. The damage done to the organ of hearing by battle noises was also under his scrutiny and a careful analysis was made by Hoople, a final report of which will be forthcoming.

Working independently and extending his field of interest to the prevention of acoustic trauma, Major Myron Hipskind took advantage of opportunities available in one of the large Paris hospitals to make very careful observations of several thousand battle casualties who had been subjected to acoustic trauma. He found the same damage to high frequency perception as did Hoople. By means of an experimental approach he showed the lack of protection to ears of the American, allied and enemy steel helmets and recommended a type helmet which would materially prevent damage to the ears. His presentation is now in The Surgeon General's office for consideration in cooperation with the research on this subject.

A very fine piece of work on battle wounds was done by Major Frank Lathrop, Major Gordon Hoople and Captain Albert C. Johnson. Lathrop recognized the possibilities for repair of facial nerves in lesions of the ear, face and mastoids. Severe lacerations of these wounds had been thought to preclude nerve suture or graft, but the diligent and painstaking efforts of these three officers proved beyond doubt that an anatomical restoration of continuity was possible in a large proportion of severed facial nerves. By careful dissection beyond the region of the damage, the nerve trunks were found, sutured

by rerouting if necessary, or grafted with fresh pieces from the femoral cutaneous nerve. Grafts from 8 mm. to 55 mm. were placed. Approximately 50 cases were so treated before return of the soldier to the U. S. A. The nerve registry follow-up system of The Surgeon General's Office will give the final information. Encouragement for the method of grafting this particular nerve comes from a recent report given at the British neurosurgical meeting in June 1945, at which Professor Norman Dott demonstrated a case of facial nerve graft, 15 cm. in length, following removal of an acoustic tumor, with excellent clinical results. The results of Lathrop, Hoople and Johnson in this series should provide added evidence for the long-held opinion that the facial nerve has excellent powers of regeneration and is not comparable to other peripheral nerves where the results are not so encouraging. Hypoglossal and spinal accessory anastomoses should not be done until a thorough trial of direct central and peripheral end connections has failed. There are many facial nerve lesions in returning veterans and until the result of these early cases is definite, no other method of repair is justified.

Otolaryngology has shared in the beneficial effects of chemotherapy during the war. Control groups of cases of the common upper respiratory infections are exceedingly difficult to manage in an active theater of military operation. The Air Force carried on research with small doses of sulfadiazine in a group of airmen for prevention of respiratory infection. Preventive chemotherapy for upper respiratory infections was not used on any large group of Ground Force personnel.

Hearing Aids in the European Theater of Operations. Although the European Theater was considered one of military operation, the conditions under which the Army lived much of the time were exceedingly good. This permitted policies for service groups comparable to those in the United States. With the approval of the Theater Surgeon, electric hearing aids were supplied and issued, first to key personnel and later to any soldier whose hearing was depressed enough to affect the performance of his duty. The conservation of manpower was so emphatically urged by all Army authorities, that it was obvious that certain duties would not be hampered by the use of an aid, provided the man was psychologically adaptable. Key staff personnel were first fitted and the instruments were enthusiastically received. The Western Electric Hearing Aid was chosen to test the policy and through the cooperation of their London office, their experts fitted the aids and arranged for the preparation of individually fitted earpieces.

After success was achieved with about 30 cases of this type, the feasibility of this policy was proved. Plans were then made for the extension of the issue through selected otologists of general hospitals. Following many months of administrative and shipping delay, a sufficient quantity of Zenith No. A2A hearing aids was available to about 15 general hospitals. Between March 1 and July 1, 1945, approximately 200 of these aids were issued. Follow-up letters showed about 75 per cent satisfaction of the users. The program was not a complete success. The Zenith aids arrived too near VE-Day to work out all the details of smooth and efficient methods of distribution and battery supply. However, it was proved that for the proper patients the present elected hearing aids did increase efficiency of individuals and there is no reason why in any theater of operation they should not be used by noncombat troops. Psychological rehabilitation in those satisfactorily fitted with aids was apparent in many cases, and those who had a desire to do their job in spite of their handicap attest to the benefit derived.

It was hoped that aids could be used early for the recent cases of deafness caused by battle trauma, in order to shorten the deaf period. However, this phase of the program was never well established and conclusions as to how soon after deafness occurred an aid could be used, were not definite. Anxiety on the part of the soldiers, rapid recovery of the hearing function, and frequent moves from one hospital to another where aids were not an item of issue interfered with collecting information on this phase of the investigation.

Air Force Otolaryngology. Except for minor otolaryngologic conditions in airmen which were cared for by the flight and station surgeons, the bulk of treatments were done in Ground Force hospitals and clinics. Individual liaison between Air Force and Ground Force otolaryngologic officers was continually encouraged and some of the Air Force officers with previous specialty training did avail themselves of the opportunities of working in hospitals to which they sent their patients. Problems related to aero-otitis media were a frequent source of consideration and on the basis of previous experience with the radiation treatment, as originally advocated by Crowe, et al, this therapy was available to Air Force personnel and others where it could be advantageously applied. Largely due to the efforts of Major E. P. Fowler, Jr., radon and radium were procured and men trained to apply the treatment. Results were encouraging and on the basis of reports sent to The Surgeon General, the Air Surgeon in late 1944 sent three officers trained in the United States to England for the sole purpose of expanding the program. Treat-

ments were given at Air Force dispensaries as well as in hospitals, by the same technique as Fowler used, except that the radium was filtered through Monel metal instead of platinum. This decreased the treatment time from approximately one hour to six minutes and forty seconds. Fowler's report was recently published. The Air Force report is in the process of preparation.

Other problems of aviation otolaryngology received special attention. The availability of a pressure chamber in the Central Medical Establishment of the Air Force provided a means of detecting aero-sinusitis and loss of hearing at reduced pressures.

In cooperation with one of our colleagues of the Royal Air Force, Wing Commander Geoffrey Bateman, it was demonstrated that reduced intratympanic pressure could be instantaneously relieved by myringo-puncture without sequelae before the development of the late pathologic changes inherent in delayed readjusting of the tympanic pressure. This did not have much value in military aviation, because air crews were well trained in matters of pressure adjustment. However, the value of the treatment will probably increase as civil aviation increases, where the casual flyer is not aware of the methods used by the trained aviator. Already such cases have been reported in military personnel on leave, who have been traveling by air to recreational areas in Europe. They land with their intratympanic pressure so reduced that, unless promptly relieved, they pass through the usual stages of edema, exudation and hemorrhage inherent in tissues subjected to prolonged baratrauma.

Specialty Training in the European Theater of Operations. According to the policy of the theater surgeon no specialists were to be trained and no officers who had not already had some specialty instruction were to become specialists by virtue of their Army experience. This was for the protection of the American soldier, so that he would not be subjected to untrained specialty skill. Looking back, however, and considering that the supply of trained specialists was to be exhausted long before the hospitals sent overseas were fully staffed, a system of training interested men could well have supplied some of the deficiencies. The Army does not require, nor does it need, the best trained men in every hospital because the policy of moving patients to the specialists is now thoroughly established. What the Army did need was many more less well-trained otolaryngologists whose ability would have well cared for about 80 per cent of cases coming to the hospitals, the others being cared for by the consultation system which worked so well during the time when hospitals were concentrated in England. If the officers could not have

been supplied by the civilian population, a training program initiated by the Army in the years 1943 and 1944 would have provided better service for the troops overseas.

Soldiers Returning to the U. S. A. As the soldiers return to the United States, in addition to the functional neuroses referred to the ears, nose and throat, sinuses and larynx, there will be five classes of patients who will need special attention.

The first of this group will be the deaf. Three general hospitals are now designated for the care and rehabilitation of these patients and they will get preliminary advice and treatment before they pass into the care of the Veterans Administration. There will be a continual source of necessary consideration and a lifelong program should provide for their needs.

The second group will be the men with injured larynges whose phonatory and breathing apparatus have been damaged by wounds. Voice training and esophageal phonation will be required by some, a reconstruction of the airway by others. Plastic repair of the neck and lower jaw will enter the picture and constitute an important phase of the final rehabilitation.

Next will be the damaged facial nerves in regard to which the excellent work of Lathrop and others mentioned above will play a prominent role. In association with plastic repair of the face the physiological restoration of the facial nerve will demand the utmost care for the best results. Not until the course of the nerve has been completely explored and a graft given a chance, can it be agreed that one must be satisfied with other methods of treatment.

Chronic suppurative otitis media will comprise a large group. Many soldiers were sent overseas with this condition and due to military operations, the masking effects of chemotherapy and treatment by non-specialists, many more cases will have developed and will pass over into the Veterans Administration for their final care.

A fifth group will be those whose symptoms are due to chronic paranasal sinusitis. Likewise many went to the war with lesions in varying stages of development. Many improved during their duty abroad, others became worse and if injured in the face the infection frequently flared up with renewed and increased severity. Many sinuses have been distorted and the wound caused by apparently innoxious foreign bodies will be etiologically important in future infection.

History of Otolaryngology. Material for a fuller and more complete history of otolaryngology in World War II is now being collected by The Surgeon General's Office. This will include not only a statistical analysis of several phases of the specialty, but will contain contributions by many men who actually did the work with patients. Too high a tribute cannot be paid to the diligent, alert, and well-trained men who provided the large proportion of otolaryngologic care to the European troops. Working under the conditions of war the best policies which govern our peacetime decisions are not possible. The Army rightfully demands men for active duty and the greatest number who can be on duty constitute the Army ideal from its Medical Department. The casualty is a detriment to the advancing Army and because the enemy is frequently such an unknown factor, thereby disrupting all plans for orderly progress, the sick and wounded must be a secondary consideration to the main end in view.

The civilian population has placed the Army in the field for the protection of its way of life. Civilians have the power collectively to determine Army policy. Civilians had the privilege of knowing what the Army was doing during peacetime to prepare for war, and if the medical care provided by the Army during wartime was creditable, the civilian population was responsible. If the service provided was not of the type desired for the care of the families and friends of civilians, it behooves them at once to spend more time and effort to make it so. Specialty medical care during war will depend upon the attention given during peace to laying the necessary foundation for wartime specialty care of the soldier. Legislative pressure is still the dominant force in our democracy and when effectively applied does produce results. As a significant group of doctors the otolaryngologists have not been particularly active in this respect, and as a group of specialists for future contribution to human medicine, the time is ripe for reflective contemplation and active participation.

OFFICE OF THE SURGEON GENERAL
WASHINGTON, D. C.

X

AN OTOLARYNGOLOGIST'S EXPERIENCE IN WORLD WAR II

GORDON D. HOOPLE, M.D.

SYRACUSE, N. Y.

This is not a scientific paper. It is the story of one otolaryngologist's career during the late war. This tale differs from that which might be told by many otolaryngologists who played a part in winning the war, for my exerience was not an average one.

I was a member of the 52nd General Hospital. This was an affiliated unit. Its staff was chosen from the faculty of the Medical College of Syracuse University. There were half a dozen men in the group with whom I had worked medically for more than 25 years. My entire medical endeavor was spent with this group. From time to time there were personnel changes in the unit but my relationship remained fixed.

I did no medical work in the United States. I joined the 52nd General Hospital two months after its activation, just two days before it moved from Camp Livingston in Louisiana to its embarkation camp in New Jersey. This was in November 1942. An unusually lengthy stay in the port area postponed our sailing date for England to the very early part of January 1943. This period was spent just waiting. Those of you who have had connection with the Army know what that means. After a five weeks' stay in a staging area in England, the 52nd moved to Wolverly in Worcestershire where it occupied a group of some 50 brick buildings and 70 Nissen huts. We worked in the former and lived in the latter for the remainder of the war. We were never moved. When I returned to the States after the fighting was over, my entire time was spent in disembarkation, recuperation leave, travel to reassembly point, travel back to separation center and terminal leave. Thus all my medical practice was done in England. This, I know, was unusual, and for me profitable.

Another personally profitable fact was this. My medical work was entirely otolaryngologic. This is in sharp contrast to what hap-

Read before the Eastern Section of the American Rhinological, Laryngological and Otological Society, Boston, Mass., January 9, 1946.

pened to some otolaryngologists. My administrative duties were negligible.

Ours was the only otolaryngologic department in the European Theatre of Operations (out of several hundred) with at least two men on service during the entire period of the war. At intervals there were three. In all, 13 men were assigned to the department. They had varying degrees of training and experience. The service was active enough to afford good teaching facilities. There were nearly 10,000 new patients and over 43,000 clinic visits. In fact, except for the absence of a pediatric service, the requirements for a good residency were at hand. Inasmuch as we were active for nearly a year and a half before the invasion took place, the service could have trained several men. In reality, the service did considerable teaching, even though, of the 13 men mentioned above, only one was permitted to remain with us longer than four months.

The work of the service divided itself naturally into two periods; pre- and post-D-Day. The hospital was surrounded by an ever-increasing number of soldiers prior to the invasion. None of the regimental or battalion surgeons had facilities to set up their dispensaries so the 52nd was designated a station as well as a general hospital.

The English are given to understatements. I think it is an understatement to say that the American nose did not do well in the English climate. Fifty-five per cent of our own command were hospitalized for respiratory infections within three weeks of our arrival in England. Other outfits did better than this but all suffered some respiratory casualties. The character of the work before D-Day then was concerned chiefly with respiratory infection complications, with the ordinary run of otolaryngologic complaints thrown in. The respiratory infections in some instances were severe. They were responsible for 26 simple mastoidectomies in the month of March 1944. These operations were necessary despite the fact that each of the 26 patients had sulfonamide therapy, the great majority of them in adequate dosage. A few had penicillin. One patient had a masked mastoiditis with the use of penicillin. It continued undiscovered till a brain abscess had developed.

In all my otolaryngologic experience I have not seen cases of acute and subacute frontal sinusitis in such a great number. Why this was so I cannot say. Others reported to me a similar experience. Even with a background of conservatism for frontal sinus surgery, I found it necessary to perform radical operations on a goodly number of these patients. One day a soldier reported to our clinic. He told

the story of the daily appearance, for seven months, of a swelling in his forehead. After a night's sleep it would disappear only to reappear the following noon. He was seen in the morning hours and his tale was questioned for no swelling was present. "If I can come back this afternoon I'll show it to you," he said. It was there when he was examined later in the day; a fluctuant swelling over the left eye. It proved on operation to be a subperiosteal abscess which was connected with the frontal sinus by an osteomyelitic fistula of the anterior wall of the frontal. This is one example of many bizarre cases seen.

I have long had difficulty in maintaining a patent nasofrontal passage following radical procedures on the frontal sinus. We were taught early in our experience that acrylic, a plastic, could be used in apposition to mucous membrane without exciting reaction in the latter. All of our later radical operative procedures on the frontal included the following technique. A short, properly molded tube of acrylic, surrounded with a skin graft, was placed in the nasofrontal opening and allowed to remain in place as long as desired. This proved much more satisfactory than any procedure I had previously used. In one soldier, a bleeder, the tube was left in situ for three months. We were particularly desirous in this case to obtain patency in order to avoid further surgery because of the blood dyscrasia. At no time during the three months was there any swelling surrounding the tube nor did it cause any ulceration. The cleanliness of the part was remarkable. It is not my intention ever again to use rubber tubing in the nose.

Another very prevalent condition was acute catarrhal otitis media with fluid or acute secretory otitis media as it is known in Boston. It was just as difficult to diagnose as it is in civilian practice and even more difficult to cure. The duration of the history in some of these was amazing. A large experience with this condition prompted me, when I was asked to read a paper before the Royal Society of Medicine, in London, to choose a discussion of this topic. Dr. Mosher, when I returned to this country, dryly remarked, "You chose a subject about which you knew nothing." I agreed that this was probably true at least as far as treatment was concerned. However, I found very few men, even those well trained in otolaryngology, who could properly diagnose acute catarrhal otitis media with fluid. I still believe it is the oftenest missed diagnosis in otology.

Still another prevalent ante-D-Day condition was fractures of the face. There was an outstanding contributing cause—the blackout. When a touch or two of spiritus frumenti was added, the result-

ing calamities were additionally serious. The frequent use of the bicycle by inexperienced riders was another and not infrequent cause. A number of these fractures were complicated by skull fractures. The British had much experience with this with their previous four years of the blackout. They warned us to be watchful of dural tears along the skull fracture line. Their experience had taught them that surgical repair of the tears (usually with fascia lata grafts) was a preventive of subsequent meningitis or brain abscess. Several of these were discovered and operated upon by our neurosurgeon. Reduction of a superior maxillary deformity through an intranasal opening into the antrum by means of a curved blunt elevator proved much more satisfactory in our hands than any attempts to elevate depressions by hooks. A few open reductions were necessary, though in the main such procedures were discouraged. These were in the plastic surgery category and patients who needed plastic surgery in the main were shipped home. Plastic procedures take time, and hospital beds in a theatre of operations must be available for battle casualties. This was a note constantly sounded and heed was paid it.

Army life as affected by war was responsible for some of the prevalencies. I cite two examples. Otomycosis was a constant annoyance, both to the patients and the doctors. How did our soldiers get otomycosis? They were, most of them, trained in the South where they became hosts to the fungi and then were shipped to England. Why the Army put boys in our Southland and then transferred them to the cold damp climate of England I could never understand.

Allergies were a bugbear. They are in civilian practice; they were more so in the Army. I feel there were three factors in their prevalence. One was the introduction to the mildly allergic of new and exciting allergens; second, the dusty atmosphere of barracks and quarters heated by small stoves with the frequent application of new coal and the removal of ashes; and, third, the fact that Army life with all its frustrations, separations, and fears is as potent as anything I know for unbalancing the autonomic nervous system.

When we came to the post-D-Day period the character of our work changed materially. By this time most of the soldiers had become acclimated, respiratory infections had subsided and the ever-increasing numbers which landed on the continent diminished our surrounding personnel. We were left free to care for casualties.

An outstanding recollection of this period was the arrival of hospital trains, of which the 52nd General Hospital had a greater

number than any other similar unit in England. The transportation corps of the Army did an outstanding job in this war and the conveyance of the wounded was no exception. The casualties arrived in trains which carried approximately 300. Though the railroad station was better than two miles from the hospital, the 300 would often be in their beds within an hour of their arrival. Though their wounds were severe and extensive, trainload after trainload would arrive without a notable elevation of any soldier's temperature. This is a tribute to the efficient administration of penicillin and the sulfonamides. It was really remarkable. Secondary suturing was possible in the very great majority of instances the day after arrival.

For an otolaryngologist, some of the most interesting patients were those whose sinuses, ears or necks had been pierced by one or more foreign bodies, the most frequent of which were shrapnel fragments. How some escaped death could not be imagined. I made the following statement at the time and I have had no cause to change it. "We see the miracles; the dead are on the beachheads and in the fields." It was soon learned in this group of patients that other foreign bodies than those primarily responsible for the injury might be lodged in the wound or in one of the sinuses. These might be pieces of wood or clothing and, because of the lack of x-ray evidence, be unsuspected. In several of the earlier cases there was continued suppuration from an external wound or a sinus, which upon re-operation was found to be due to such a cause. It soon became the rule, and apparently a wise one, that we were to operate on and thus exteriorize every sinus which had been entered by a foreign body. Here again, the frontal sinus was the worst offender, undoubtedly because of its narrow nasofrontal duct. Left unoperated, these very frequently continued to suppurate through the wound of entry. Exploration, without surgical manipulation of the duct, in almost every case resulted in cure. Closure of the wound was allowed when the duct showed itself to be patent by the method of instillation of saline into the sinus with subsequent sensations of its presence in the nose or throat.

An interesting and challenging number of such patients were those in which the foreign body, after passing through some portion of the face, finally lodged in the region of the pterygomaxillary fossa. This always caused a trismus which seemed not to be relieved till the foreign body was removed. The most successful approach in these patients was the transantral route with or without ligation of the external carotid artery. Localization was not easy in this space and on more than one occasion resort had to be made to localizing x-rays with markers in place in the midst of an operation.

Included in a very large group were those whose auditory apparatus suffered injury through blast. A fair number of these had ruptured membrana tympani. It was impossible to determine why some had this complication and others did not. Profound and irreparable deafness often followed blast with no evidence of rupture present. Secondary infection following rupture was infrequent. When present, it rarely required subsequent mastoidectomy. It is interesting to note that if suppuration followed rupture, it was the rule that with the cessation of the discharge, the drums would heal. The opposite was not necessarily true. Most ruptures healed spontaneously, but a fair number did not. If the tear extended to the annulus, it seemed impossible to bring healing about. However, with proper care, even though the perforation was large and the lower end of the malleus in its normal location lay free of drum attachments, if there was marginal drum tissue, healing was brought about. The judicious use of protheses or splints we found to be the most useful adjuvant. Very few patients with unhealed drums left the 52nd. A study was made of cochlea injury due to blast of soldiers who did not complain of hearing loss. Twelve hundred audiograms were taken on such patients. These produced evidence that there are many unsuspected instances of auditory injury due to this cause. This work will be reported at a later time.

Late in the war, in fact in the spring of 1945, a directive came out of headquarters which stated that injuries to the facial nerve, except those in the peripheral portion, were to be treated by certain designated otologists. This was prompted in part by the good results obtained by Major Frank Lathrop in a large series. I was permitted to participate in the program though to a limited degree because of the lateness of its institution. In the majority of these end-to-end suture was possible, but, in a few, nerve grafts, usually from the lateral cutaneous nerve of the thigh, were used to restore continuity. This was exceedingly interesting work and furnished the high point of my surgical endeavors.

Another role to which I was assigned and which was most enjoyable was that of consultant. The hospitals of the European Theatre were divided geographically into hospital centers and consultant otolaryngologists were appointed for each center. I was given one of these assignments. It happened that the 52nd was located in the most populous center of all and I had the pleasant task of frequent visits to 19 hospitals. Some very interesting cases were seen and many very pleasant relationships established. In the fall of 1944, this assignment was extended in scope when I was asked to sub-

stitute for the Senior Consultant in Otolaryngology, Colonel Norton Canfield, while he was on leave. This duty took me to France for a stretch of nearly three weeks. Approximately half of this time was spent in the Paris area, while the other half was taken up with a tour of the Cherbourg peninsula where I visited many of the General Hospitals then stationed there.

I cannot close this narration without a tribute to the British people. I came to know many of them exceedingly well and profited personally by this association. I saw their deprivations and believe me they were considerable in number and variety. I visited most of the bombed cities and the destruction was awful. I noted their stalwartness and courage, without which the Battle of Britain would not have been won and the whole course of the war would have been different. The British otolaryngologists were most hospitable. We were welcomed at all their meetings and this last June at their annual banquet all available American otolaryngologists were their guests. Many of us were personal guests in their homes and in their hospitals and can never forget the cordial relations they established.

This is a simple story, but for me, it was an unusual experience. Like you, I rejoice that it is over and we can, once more, return to our normal endeavors.

XI

PROGRESS IN OTOLARYNGOLOGY SINCE 1937. AN APPRAISAL OF THE PRESENT STATUS OF DR. MOSHER'S "UNSOLVED PROBLEMS"

FREDERICK T. HILL, M.D.

WATERVILLE, MAINE

In his address as the Guest of Honor of the American Academy of Ophthalmology and Otolaryngology in 1937, Dr. Mosher chose as his subject, "Unsolved Problems in Otolaryngology." This paper was typically "Mosheresque." While necessarily covering much ground, so that the author termed it "staccato in effect," it did so systematically. It was frank, searching and based upon a rich experience, both in research and in teaching. It was punctuated by those personal observations which are so characteristic. And it was concluded by several paragraphs of priceless philosophy.

By way of introduction Dr. Mosher referred to things he had seen solved. Among these he included pulsion diverticulum of the esophagus, chronic dacryocystitis, cardiospasm, or as he prefers, fibrosis of the terminal portion of the esophagus, deep cervical abscess, and the surgical management of cancer of the larynx. Then he posed his challenge in the Unsolved Problems. At this time it would seem opportune to ask ourselves what progress had been made toward the solution of these problems.

Dr. Mosher listed as unsolved problems: chronic infection of nasal and pharyngeal mucous membrane, focal infections, anaphylaxis and allergy, cancer of the esophagus, osteomyelitis of the frontal bone, orbital abscess, the radical frontal sinus operation, atrophic rhinitis, lung abscess, bronchiectasis, acute tracheobronchitis, cavernous sinus thrombosis, chronic progressive deafness, otosclerosis, Ménière's disease, brain abscess and meningitis.

Which of these may we reasonably consider as solved to-day; or, to put it better, consider capable of solution in proper hands? This distinction is important for there will never be a universal solution for the problem of human ills. The manifold advantages of

Read before the Eastern Section Meeting of the American Laryngological, Rhinological and Otological Society, Boston, Mass., Jan. 9, 1946.

penicillin or the sulfonamides, for example, may become quite the reverse on empiric application. But assuming correct diagnosis, intelligent management and adequate treatment, in which of these conditions may we properly and confidently expect sufficiently satisfactory results to indicate a solution?

Chronic Infection of Nasal and Pharyngeal Mucous Membrane. Speaking of chronic progressive infection of the nasal and pharyngeal mucous membrane, Dr. Mosher said, "He who can arrest or cure this condition will prevent or cure chronic progressive deafness." He could well have added an allied problem, that of hypertrophy of the lymphoid structures of Waldeyer's ring. Both of these remain unsolved problems, possibly metabolic in origin. Clark's[1] paper on the relation of the size of tonsils to diet, published in 1923, indicated that a high fat and high calorie diet caused an increase in size of the lymphoid nodules, both in the tonsil and in Peyer's patches. Crowe's work on the radon treatment of nasopharyngeal lymphoid tissue has received considerable impetus through its employment in aviation medicine during the war and undoubtedly has wider application in the future. But in the main we still are faced with unsolved problems. At least we are no nearer in curing chronic progressive deafness, although we may feel that we sometimes prevent it by proper management of lymphoid hypertrophies in early childhood.

Focal Infection. Focal infection has suffered from overplaying. Operating upon some possible focus just because it is easily visible and accessible, while neglecting some equally possible but more remote location, has done much to bring the theory of focal infection into disrepute. I think, however, that we see less people bereft of teeth, tonsils and mucous membrane than formerly and that a saner, more conservative attitude pertains. We hope, at least, that remunerative chronic tonsillitis is on the wane.

Allergy. The allergist has come into his own as a full-fledged specialist and, if given the chance, can prove a beneficent influence on the over-zealous rhinological operator. It is still appalling to see the number of cases in which radical surgery is advised without any consideration of the possibility of allergy. Careful histories and repeated nasal smears will avoid many pitfalls. The routine office use of the Hansel stain is very much worthwhile. In this I would say we had made progress but much more was indicated.

Cancer of the Esophagus. Dr. Mosher expressed a ray of hope regarding cancer of the esophagus. This hope has been realized in the work of Sweet[2] who has solved the problem of carcinoma of the

lower two-thirds of the esophagus, a solution better than Mosher hoped for. His high esophagogastric anastomosis frees the stomach sufficiently to bring the fundus to the level of the aortic arch, or when necessary, above it, in which case the upper end of the esophagus is mobilized and pulled up in front of the arch.[3] He has done 25 such operations with eight deaths, a mortality of 32 per cent.* This must be classed as an outstanding achievement. Lateral pharyngotomy, as developed by Orton in this country, provides a surgical solution for certain of the upper esophageal malignancies. At the same time improved roentgen technique offers greater hope of palliation, if not cure.

Osteomyelitis of the Frontal Bone. Dr. Mosher had very largely solved the surgical problem of osteomyelitis of the frontal bone. It only remained for the development of the sulfonamides, and later of penicillin, to complete the chapter and in most cases at least minimize, if not prevent, the extensive loss of bone previously necessary. His paper stressed the need for careful diagnosis, differentiating between the fulminating and the sequestrating types, and called for the best judgment in individualization of each case. His surgical principles still hold, only simplified by chemotherapy and minimized as to final results by the use of bone grafts or vitallium plates.

Radical Frontal Operation. The radical frontal sinus operation is still an unsolved problem. Obliteration of the sinus has the objection of resulting deformity. Epidermatizing the enlarged nasofrontal duct looks better in print than in the nose. Goodale's recent work with tantalum foil may prove an effective answer. Similarly the ethmoid operation, whether external or intranasal, is often far from perfect in its result. Perhaps it is too much to expect completely successful surgery in the ethmoid labyrinth where scar tissue may continue to obstruct drainage. Too frequently little or no attention is paid to the physiology of these sinuses. Van Alyea[4] states

*Dr. Sweet's operative record, revised up to October 30, 1945, is of such interest that I have requested his permission to include it in this paper.

	NO. CASES	DEATHS	MORTALITY
Transthoracic total gastrectomy with esophagojejunostomy in the chest.	21	7	33%
Carcinoma of the cardia arising either in the esophagus or in the stomach requiring resection of the stomach and esophagus with low esophagogastric anastomosis.	56	8	14.2%
Carcinoma of the midthoracic esophagus with high esophagogastric anastomosis, many of them above the aortic arch.	25	8	32%

that "In the treatment of nasal conditions the preservation of functioning structures is mandatory." In those cases when, after careful study, radical operation is deemed necessary, it should be understood that the result is apt to be a poor substitute for a normal nose, and the decision for operation made only when this possibly poor result will be better than what can be expected from more conservative measures. The problem of orbital abscess can be considered as solved, at least if the ophthalmologist has the advantage of an otolaryngological consultation.

Atrophic Rhinitis. Atrophic rhinitis is still a problem and threatens to remain so until it disappears from our midst. Estrogenic therapy is helpful in keeping the condition under control, but we have no cure nor do we know the cause of this disease. Whether this is endocrine or genetic, as suggested by its prevalence in family groups, is a question; but its apparent gradual disappearance, at least in New England, points to the latter supposition, as there seems to be little decrease in endocrine disturbances among our female population.

Acute Tracheobronchitis. The problem of acute tracheobronchitis has been more nearly solved by a combination of definitive bacteriology, chemotherapy, humidification, oxygen, convalescent serum and intravenous fluids, together with tracheotomy and bronchoscopic aspiration when necessary. Baum's[5] suggestion of intravenous concentrated plasma to lessen subglottic edema seems worthwhile. There is still need for extreme vigilance in the care of these cases if tragedy is to be avoided.

Lung Abscess and Bronchiectasis. Through the combined efforts of the chest physician and the thoracic surgeon, assisted by the bronchoscopist, lung abscess and bronchiectasis present much more satisfactory problems than formerly. The local use of penicillin together with bronchoscopic aspiration is quite effective in certain pulmonary abscesses, while others require thoracic surgery. One of the bright spots in the picture is the employment of lobectomy in bronchiectasis. Even with involvement of both lungs, removal of the most affected lobe, or even lobes, may exert favorable influences on less involved areas.

Cavernous Sinus Thrombosis. Cavernous sinus thrombosis is no longer the hopeless proposition it once was, due again to chemotherapy, supplemented by heparin to prevent clot formation. Brilliant results have been reported by Schall[6] and others. This has made unnecessary the extensive orbital operation formerly suggested by Dr. Mosher.

Meningitis. Likewise, meningitis, designated by Dr. Mosher as "one of the chief, if not the chief, of unsolved otological problems," has succumbed to modern chemotherapy. In his paper, eight years ago, he strongly hinted at the solution with an observation on the then new use of sulfonamide derivatives (prontosil and prontylin). Eradication of the infection in the mastoid, together with adequate sulfonamide, or penicillin therapy, based upon definitive bacteriology, and lumbar punctures as needed, have changed our conception of this formerly hopeless complication.

Labyrinthitis. Labyrinthitis continues to present a problem, although of less frequent occurrence because of the better management of otitis media. The Bárány tests are not infallible and diagnosis is often difficult, except in the case of diffuse suppurative labyrinthitis. It is well to keep in mind Dr. Mosher's observation that "many a chronic ear with labyrinthine symptoms lost them after a radical mastoid operation." Recently Jones[7] has advocated the endaural approach in these cases in order to reach the perilabyrinthine cells not accessible through the posterior incision. In the case of diffuse suppurative labyrinthitis the dictum of labyrinth operation upon a rising cell count in the spinal fluid still holds as it did in 1937.

Brain Abscess. The problem of brain abscess remains very much as stated by Dr. Mosher, "first to find it, and then to drain it successfully." Localization still presents the difficulties it did eight years ago, sometimes relatively easy, many times difficult. Chemotherapy, both locally and parenterally, has been a valuable adjunct once drainage has been established.

Petrositis. Petrositis, like labyrinthitis, is encountered less frequently, due to the greater prevention of complications with chemotherapy. The endaural operation seems to offer many advantages, although the temporal fossa approach may be extremely useful.

Facial Nerve Paralysis. Dr. Mosher considered the problem of paralysis of the facial nerve nearly solved by the investigations of Ballance and Duel. The work of Tickle with nerve transplants and of Furstenberg with end-to-end anastomosis would seem to remove all doubt as to the solution of this problem.

Ménière's Disease. Dr. Mosher stated that the problem of Ménière's disease was on the way toward surgical solution through the operation of section of the vestibular portion of the eighth nerve. However, this does not control the degenerative process in the internal ear. More recently Day has developed electrocoagulation of the

labyrinth through the uncapped horizontal semicircular canal. Both of these procedures are rather formidable and would seem applicable only in the extreme case. This condition is probably vasomotor in origin. Mogan and Baumgartner[8] feel that it is similar to Raynaud's disease or migraine, and report a case relieved by cervical sympathectomy. Hallpike's[9] observations suggest that it is due to obstructive distention of the endolymphatic system. If so, a revival of Portmann's operation on the saccus endolymphaticus may be forthcoming. Williams[10] prefers the term "endolymphatic hydrops." He feels that it is a form of physical allergy and uses nicotinic acid in treatment, for its vasodilating effect. Shelden and Horton[11] have reported excellent results from histamine therapy, while the Furstenberg treatment, or the simpler potassium chloride medication has had wide acceptance. Until more is known as to the exact cause, Ménière's disease must be considered as an unsolved problem.

Otosclerosis. The greatest advance in the treatment of deafness has been in the surgical management of otosclerosis by fenestration. Lempert's[12] series of 1000 patients, with 57 per cent completely rehabilitated by restoration of hearing and 9 per cent more, partially rehabilitated, is most impressive. By this procedure clinical otosclerosis may be converted into symptom-free otosclerosis. It is not a cure for the disease and the cause of otosclerosis is still an unsolved problem, but it treatment is no longer "the great field of the quack." It is not too much to expect that continued observations will confirm or still further improve the present technique and suggest more accurate criteria as to cases favorable for operation. It is also to be hoped that this operation will not suffer from too much lay publicity and possible exploitation of the credulous through too widespread and perhaps inexpert employment, such as to detract from the credit rightfully due Lempert.

In the treatment of deafness, other than otosclerosis suitable for fenestration, whether conduction or perception, the problem is still one of amplification, education and rehabilitation. Improvements have been made in hearing aids and greater attention devoted to proper selection, but the problem is far from solved. Now that the war is over, we may begin to look for more tangible results from the work of the Academy Committee on the Conservation of Hearing.

Referring to the physiology of the ear, Dr. Mosher suggested that the many papers which followed the discovery of the Wever-Bray phenomenon had caused dyspnea, if not anoxemia among listening otologists. But out of this has come a much better conception of auditory function while the exigencies of war have stimulated

valuable investigations by Lurie and others as to the disastrous effects of excessive noise upon the cochlea.

In his paper Dr. Mosher interpolated a paragraph on the training of the specialist. This is still a problem, brought to the fore today by the deficiencies of the accelerated program of medical education and the demands of many of our medical veterans for further training. It is doubtful if sufficient desirable opportunities for training in otolaryngology can be provided through residencies alone and some supplementary system, such as preceptorships, may be necessary. In all this discussion one thing should be kept in mind. That is, that no one's education is ever complete and that a constantly pursued program of continuation education is more important than the best residency, if the latter is followed by a sense of complete self-satisfaction and mental stagnation. Every effort must be made to provide the best possible training, in residency when possible, by basic courses and preceptorships when necessary, but this will lose its greatest value if it does not stimulate the desire for constant improvement afterward. Every hospital should provide opportunities for continuation education and make participation a requisite for staff membership.

SUMMARY

To summarize, one might try to strike a balance of the present status of Dr. Mosher's list of Unsolved Problems of 1937. On the credit side one might place orbital abscess, osteomyelitis of the frontal bone, cancer of the esophagus, acute tracheobronchitis, lung abscess, bronchiectasis, cavernous sinus thrombosis, meningitis, facial paralysis and the surgical management of otosclerosis. On the debit side one must list chronic infection of the nasal and pharyngeal mucous membrane, focal infection, the radical frontal and ethmoid operation, allergy, atrophic rhinitis, brain abscess, Ménière's disease, and the prevention and amelioration of progressive deafness, although encouraging progress can be claimed for many of these. If one might hazard a guess as to the future, further progress will come with a better understanding of the etiological factors in these conditions and by greater attention to the physiology of the structures involved. Much will depend upon studies, such as those of Proetz upon the nose and sinuses, and of Lurie on the ear. But, in the future, as in the past, there need be no excuse for an Alexandrian attitude. There will be no paucity of problems to solve.

THAYER HOSPITAL.

REFERENCES

1. Clark: Studies on the Relation of the Size of Palatine and Pharyngeal Tonsils to Diet, Trans. Am. Laryng., Rhin. & Otol. Soc., 1923.

2. Sweet, R. H.: Surgical Management of Carcinoma of the Mid-Thoracic Esophagus, New England J. M., Vol. 233, July, 1945.

3. Sweet, R. H.: Personal communication.

4. Van Alyea, O. E.: Management of Chronic Sinusitis, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 54:443 (Sept.) 1945.

5. Baum, H. L.: Treatment of Acute Laryngotracheobronchitis, Arch. Otolaryng. 41:175 (March) 1945.

6. Schall, L. A.: Treatment of Septic Thrombophlebitis of the Cavernous Sinus, J. A. M. A. 117:581 (Aug. 23) 1941.

7. Jones, M. F.: Disturbed Vestibular Function: Causes and Cures, Arch. Otolaryng. 41:272 (April) 1945.

8. Mogan, R. F. and Baumgartner, C. J.: Ménière's Disease, Arch. Otolaryng. 41:113-116 (Feb.) 1945.

9. Hallpike, C. S., and Cairns, H.: Observations on Pathology of Ménière's Syndrome, J. Laryng. & Otol. 53:625-654 (Oct.) 1938.

10. Williams, H. L.: The Syndrome of Physical or Intrinsic Allergy of the Head. Ménière's Disease (Endolymphatic Hydrops), Proc. Staff Meet., Mayo Clinic 20:373 (Oct. 17) 1945.

11. Shelden, C. H., and Horton, B. T.: Treatment of Ménière's Disease with Histamine Administered Intravenously, Proc. Staff Meet., Mayo Clinic 15:17-21 (Jan. 10) 1940.

12. Lempert, J.: Lempert Fenestra Nov-Ovalis with Mobile Stopple, Arch. Otolaryng. 41:1-41 (Jan.) 1945.

XII

STATUS ASTHMATICUS

GEORGE O. CUMMINGS, M.D.

PORTLAND, MAINE

Status asthmaticus may be defined as a state of severe, unremitting asthma, which, if not relieved, results in death.

This paper falls into three parts: first, remarks concerning its pathology and pathological physiology; second, its treatment with especial reference to bronchoscopy; and third, reports of four illustrative cases.

According to Hilding,[1] the most important pathological change is the metamorphosis of ciliated epithelium to mucus-producing goblet cells. This results in the filling of the bronchioles with a thick tenacious mucus which can be removed with difficulty as the contents of some of the semi-evacuated cells is one with a mucous mass free in the bronchioles. Another equally significant concept advanced by Hilding[2] is the production of atelectasis by ciliary movement of mucus in the bronchioles. When changes occur in the character of the mucous secretion and the lumen of a bronchiole is lessened by shortening and contracting during respiration, the mucus on its side walls unites to form a diaphragm. Ciliary action moves the diaphragm until it tends to be halted by negative pressure built up from behind. The cilia, however, continue to move its margins forward as its central part thins and finally the mucous diaphragm again reforms central to its initial position. As such mucous pistons move upward, negative pressure occurs peripherally in the bronchi and atelectasis develops. At autopsy, the lungs may present gross evidence of emphysema and atelectasis and air-filled bullae may be seen on the surface.

We may believe that, as an asthmatic attack develops and progresses toward a status asthmaticus, there first occurs bronchial spasm, soon accompanied by pale edema of the submucosa, followed by metamorphosis of at first a few and then more and more ciliated cells to goblet cells. As these changes are beginning to take place the small amount of mucus first secreted begins to form piston-like dia-

Presented at the meeting of the New England Society of Otology, Rhinology and Laryngology, Oct. 3, 1945.

phragms and, as negative pressure develops, edema of the submucosa increases and the metamorphosis of ciliated to goblet cells is hastened until finally the bronchioles become filled with thick tenacious mucus. While this is occurring, the patient is experiencing increasing dyspnea. The expiratory effort is more marked, for at inspiration with the expansion of the airways many of the mucous diaphragms tend to break and permit air to enter the finer bronchioles and alveoli. Here it tends to become trapped since, as expiration proceeds with the shortening and contracting of the bronchioles, the thick tenacious mucus tends to occlude their lumens and such air as gets out is forced to pass through air passages narrowed because of the mucus.

Asthma is a disabling but rarely a killing disease. Why only a few asthmatics develop status asthmaticus is unknown, unless it is because their fundamental allergy is reinforced by bacterial or virus infection. In any event, the pathological processes in most asthmatic attacks and in many patients with status asthmaticus are reversible.

In general, many of these patients are dehydrated and lack nourishment. Infusions of a ten per cent glucose solution may be of great value. Digitalis may be necessary to aid cardiac function. The patient should be in a room free of dust, hangings and upholstered furniture. Allergen-proof casings should be used on pillows and mattresses. High humidity with average room temperature is to be desired. Good nursing is essential. In particular, the treatment of status asthmaticus is directed toward widening the airways of the bronchial tree, aiding the elimination of mucus, resting the patient with sedatives and using drugs to combat infection. In fact there are few diseases in which so many different drugs may be of real use.

Drugs Useful in Widening the Airways. This is best accomplished by epinephrine which is only of value if given parenterally. It may be given in a 1-1000 solution hypodermatically or intramuscularly in doses of from 0.2 to 0.5 cc. and is more effective if given in small doses repeated at 20 minute intervals. It can be given in similar doses intravenously very slowly in increments of from 0.05 to 0.01 cc. Epinephrine in oil takes longer to function and may relieve ordinary asthma for from 4 to 24 hours. It is given in doses of from 0.5 to 1.5 cc. intramuscularly. Epinephrine in a 1-100 solution may be sprayed directly into the larynx with a special all-glass nebulizer. It also blanches and dries the patient's throat. This may be counteracted somewhat by having the patient first swallow a small amount of glycerine.

Ephedrine and the numerous ephedrine-like substances have the advantage of being effective when given by mouth. They are sometimes combined in capsules or tablets with some of the barbiturates and other medications. They are usually not adequate in severe asthma. In aqueous solutions, they may be sprayed deep into the pharynx with a special nebulizer, into an oxygen tent or mask, or they can be dropped into the tracheobronchial tree with a laryngeal syringe.

Epinephrine and ephedrine have the disadvantage that they both cause a rise in blood pressure, so when the patient is hypertensive, the physician must decide whether the asthma or the treatment is the more harmful. The use of epinephrine and ephedrine, however, is followed by a secondary engorgement, more readily observed in the nasal mucosa than that of the tracheobronchial tree. Patients occasionally become epinephrine-fast in the course of a status asthmaticus and, as this condition resolves, epinephrine again becomes effective. Such epinephrine fastness is more apt to occur in dehydrated patients and when the glycogen stores in the liver have become depleted.

Aminophylline or theophyllin with ethylene diamine is irritating to the stomach and must be given intramuscularly, intravenously or by suppository. This is an extremely useful drug in patients refractory to epinephrine. It may be given intramuscularly in doses of 7.5 grains but is painful. It is best given slowly intravenously in doses of from 3.75 to 7.5 grains in 10 and 20 cc of distilled water respectfully.

Atropine, while tending to relieve bronchial spasm, dries secretion and is not useful in this condition.

Drugs Aiding the Elimination of Mucus. The iodides have been used for years in the treatment of asthma. Either the sodium or the potassium salts may be administered in saturated solution. The initial dose is usually 15 grains by mouth. Occasionally intravenous administration of 15 grains of sodium iodide in a 10% solution is effective. Lipiodol has been injected by means of the bronchoscope or with a laryngeal syringe with the intention of applying the solution directly to the mucosa of the tracheobronchial tree. It undoubtedly defeats its purpose by adding to the mucus already in the airways and increasing the work of the cilia. (I have had one patient develop an iodine rash as the result of the introduction of 20 cc. of lipiodol for a bronchogram.)

Easing the Entrance of Oxygen to the Alveoli. The purpose of respiration is to bring oxygen to the blood vessels in the alveoli.

Respiration is eased if the amount of oxygen in the air is increased. Therefore the patient with dyspnea does better in an oxygen tent or with an oxygen mask. The mask has the additional advantage of preventing the inhalation of any allergens that are in the air of the room. Helium has been used with oxygen as an inert gas of a lighter specific gravity than nitrogen. If helium 80% and oxygen 20% are administered in a special helium-proof tent or with a closed mask, the force necessary for drawing this mixture into the lungs is only one half that required for air.

Drugs Useful in Resting the Patient. Sedatives are of value in preventing the patient from wearing himself out and relieving his apprehension. Sometimes, after a period of sleep, there is a relaxation of the bronchial spasm. Morphine, codein, and most opium derivatives should not be used for they depress respiration, deaden the cough reflex and cause bronchiospasm. Vaughan[3] and others have reported death after the use of morphine in asthma. Sedation, in various degrees, may be accomplished by the use of one or another of the barbiturates. Sodium pentothal has been employed. Chloral hydrate is a sedative and hypnotic that warrants more frequent use. It may be given in doses of 7.5 to 15 grains either in a syrup by mouth or in olive oil as a retention enema. Paraldehyde may be given orally in doses of from 10 to 20 cc. diluted with one or two parts of olive oil. It can also be given slowly in doses of from 4 to 5 cc. intravenously, but not without some risk. It should be stopped when the desired depression is reached. Five to 20 cc. may be given intramuscularly but it is painful. Both ether in oil and avertin have been administered by rectum when patients have been refractory to other drugs. A new synthetic drug, demoral, has an antispasmodic effect on the bronchial musculature, a sedative action and is quite effective in relieving pain. It is somewhat drying to the mucous membrane and has no deadening effect on the cough reflex. Its average dose is 100 mg.

Drugs Directed Against Infection. If the status asthmaticus has been induced by a respiratory infection or if a rising temperature and blood count suggest an infection, the use of sulfonamides or penicillin may be invaluable. The use of penicillin as an aerosol introduced deep into the pharynx or into an oxygen tent or mask by means of a vaponephrin nebulizer has been suggested.[4]

Benefits of Bronchoscopy. A bronchoscopy can easily be performed on a patient in status asthmaticus with local anesthesia with little discomfort to the patient. Bronchoscopic aspiration and ventilation is obviously useless when the bronchial tree is largely filled with

thick tenacious mucus to all intent an integral part of the mucosa. It is of value when the pistons of mucus, so ably described by Hilding, can be broken by the unimpeded inrush of air through the bronchoscope as the mucus in the larger airways is being aspirated. If then, despite adequate care, medication and watchful waiting, the patient is still dyspneic, if mucus has ceased to be raised, if physical findings or x-ray films suggest areas of consolidation, the patient should have a bronchoscopy. While the tube is in place, oxygen is allowed to flow into it. After aspiration is accomplished, 2 or 3 cc. of a 1% solution of ephedrine in physiologic sodium chloride solution may be introduced into the airways. If bronchoscopy is carefully done, it will do the patient no harm and most patients will feel a distinct benefit. I am convinced that the hyperventilation is the most valuable feature of bronchoscopy. Bronchoscopy can safely be repeated as needed.

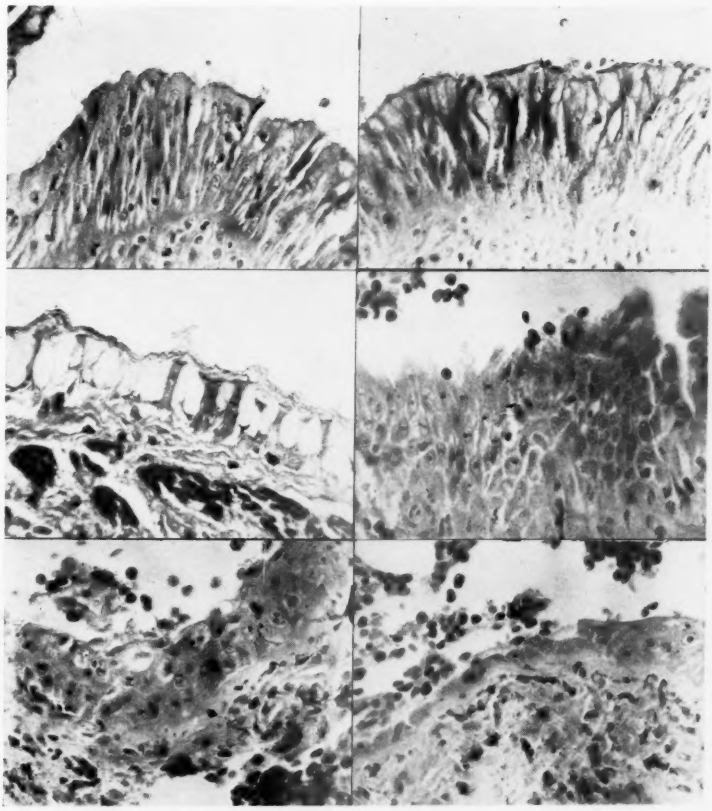
The following cases have been selected to illustrate certain facts pertaining to status asthmaticus.

REPORT OF CASES

CASE 1.—*Mild asthma for years, respiratory infection, maxillary sinusitis, status asthmaticus, medical and bronchoscopic treatment, death, autopsy findings.*

Mr. C., aged 58, had an allergic family history and, throughout his life had had asthma mildly with respiratory infections. Early in December 1942, he had a severe cold and later in the month, an x-ray examination showed an area of atelectasis at the right base. This was considered to be due to a virus pneumonia. During the next six or seven months, he worked but was not up to par physically and had a succession of respiratory infections with more or less asthma. After a vacation in August 1943, he had a respiratory infection that initiated asthma which increased in severity and during October, he received adrenalin in oil at increasing intervals. In the last week of October, he was thoroughly studied and found sensitive to dogs, cats, feathers, and house dust. Chest x-rays, at this time, were essentially negative but his antra showed retained secretion. It was felt that his asthma would clear up with attention to his antra and desensitization with house dust and autogenous vaccine.

About the first of November, his asthmatic attacks increased in severity and his physician saw him almost every other night. On November 8, he was hospitalized. His nasal mucosa had returned to normal but his dyspnea continued. The bases of both lungs were



Figs. 1-6.—A series of photomicrographs from material secured at autopsy from Case 1. These show the transition from the relatively normal ciliated columnar epithelium in Fig. 1, the beginning metamorphosis to goblet cells in Fig. 2, the almost complete metamorphosis in Fig. 3, the casting off of the remains of the goblet cells and the beginning trend toward a squamous type of epithelium with the suggestion of intercellular bridges in Fig. 4, an extention of this process in Fig. 5, to a thin squamous epithelium in Fig. 6.

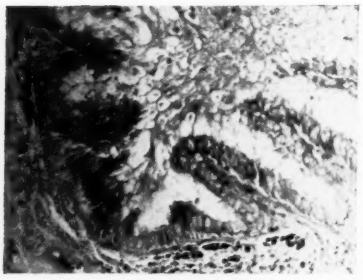


Fig. 7.—A photomicrograph from material secured at autopsy from Case 1, showing a section cut through a small bronchiole. The lining membrane has been thrown into folds by the contraction of the bronchus in an endeavor to rid itself of the mucous mass in its lumen, which to all intent and purpose is one with the goblet cells which now form its lining membrane.

flat to percussion and moist râles were heard over the rest of his chest. He raised mucus at intervals, his temperature varied little from normal, he became adrenalin fast. Aminophylline gave some relief. He was given barbiturates, paraldehyde, morphine and pantopon at times as sedatives. He was given a number of glucose infusions which contained sodium iodide. He was in an oxygen tent or used an oxygen mask a greater part of the time. Vaponephrine was used in a special nebulizer but it did not give a great deal of relief. On November 9, a bronchoscopy was done, which gave some relief. Lipiodol was injected into the airways. Another bronchoscopy was done on November 13 and a great deal of thick tenacious mucus was aspirated. He was at first relieved but soon had more difficulty, and oxygen and helium were administered. He died a few minutes later from anoxia.

Autopsy revealed that all of the smaller bronchioles and smaller bronchi in every pulmonary segment, except a portion of the right upper lobe, were occluded by bronchial casts. The larger bronchi were clear, free of secretion and unobstructed. There was atelectasis with marked reduction in pulmonary volume and actual drawing away of the lung from the chest wall for from two to four centimeters. This space was occupied by emphysematous bullae. Microscopic examination revealed considerable variations in the epithelial structure of the bronchioles. Some had their usual ciliated cells, others showed varying degrees of replacement of ciliated goblet cells,

whose contents was at times almost a part of the mucous mass in the bronchioles' lumens. In still others there was evidence of neither ciliated nor goblet cells but the lining had undergone further change and tended to resemble squamous epithelium.

Would this patient have been benefited had bronchoscopy been done earlier, his antrum infection recognized sooner and demoral (which was then not available) been used in place of morphine? Perhaps, more than anything else, if the patient could have been made to realize the seriousness of his asthma at an earlier date, the outcome might have been different.

CASE 2.—*Asthma since the age of 7, ragweed, hay fever, respiratory infection, status asthmaticus, medical and bronchoscopic treatment, recovery.*

Mrs. W., aged 22, had asthma since the age of seven. She was difficult to control and had seven admissions to the Maine General Hospital between 1940 and 1945 for severe asthma. She was sensitive to house dust, cats, feathers, orris root, and ragweed. Following fall type hay fever she acquired a respiratory infection which lit up her asthma and resulted in her admission to the hospital in status asthmaticus on December 21, 1944. She was epinephrine fast and was not relieved by aminophylline, demoral, other drugs, or the use of an oxygen tent. Physical examination suggested a patchy atelectasis and she was not raising any secretion. On December 20, a bronchoscopy was done, a small amount of thick secretion aspirated, and 4 cc. of 3% ephedrine solution instilled. She felt better at once and on return to the ward began to raise large amounts of secretion. Her improvement dated from her bronchoscopy.

CASE 3.—*Hyperesthetic rhinitis for years, asthma for three months following respiratory infection, maxillary sinusitis, status asthmaticus, medical and bronchoscopic treatment, recovery.*

Mrs. I. C., aged 40, had an allergic family history. While she did not have a pollen hay fever, she did have an occasional hyperesthetic rhinitis and stomach upsets caused by apple, celery, wheat, string beans, cauliflower, cabbage, chocolate, eggs, cats, feathers and house dust. She had never had asthma until in the fall of 1944. This followed an upper respiratory infection. On January 20, 1945, she was taken to the hospital in a definite status asthmaticus. She was raising very little secretion and physical examination suggested a patchy atelectasis. On January 21 a bronchoscopy was done, a fair amount of thick secretion aspirated and 4 cc. of 3% ephedrine sulfate solution instilled. After her bronchoscopy, her breathing was less

labored and she soon began to raise large amounts of mucus. She was epinephrine fast and at times was given aminophylline, demoral, barbiturates, luasmin, vaponephrine and oxygen. Both penicillin and sulfadiazine were used to combat infection. For six days following the bronchoscopy, she gradually improved. She had another asthmatic spell from January 27 to February 3. She again went along well until February 19, when she again had severe asthma for four days. The two last attacks yielded to medication. Pus was washed from both antra on March 1 and she was started on treatment with house dust and an autogenous vaccine.

Her recovery dated from her bronchoscopy.

CASE 4.—*Hyperesthetic rhinitis for years, intrinsic or bacterial allergy, respiratory infection, asthma for two months, status asthmaticus, unrelieved by medical treatment or bronchoscopic aspiration until nervous apprehension was relieved by intravenous luminal.*

Miss K. S., aged 41, had a mild hyperesthetic rhinitis for years. It had bothered her sufficiently so that she had nasal treatments at times during the last five years. She responded to no skin tests and probably had an intrinsic or bacterial allergy. She was admitted to the Maine General Hospital July 14, 1945, in status asthmaticus. She had a respiratory infection two months before, which was followed by increasing asthma. On admission, she was epinephrine fast. Aminophylline made her apprehensive with cold clammy extremities. Demoral gave her some relief. She also was given luasmin, coramine, infusions of 10% glucose, oxygen and helium inhalations and penicillin as her white count was 20,000 on admission. She had a productive cough the first few days of her hospital stay but then ceased raising secretion. X-ray study showed no atelectasis which was suggested by physical examination. A small amount of mucus was obtained by bronchoscopy. Her asthma was helped but little. She was extremely apprehensive and remained so until she was kept under sodium luminal given intravenously for some 12 hours. When she awakened, she was much improved. The psychic factor may be present in severe asthmatics.

SUMMARY

1. The most significant pathological factors in status asthmaticus are the metamorphosis of ciliated to goblet cells and the production of atelectasis by the advance of mucus toward the larger airways.

2. The medical treatment of this condition requires the knowledge of many drugs to widen the airways, to aid elimination of mucus, to ease the entrance of oxygen to the alveoli, to rest the patient, and to combat infection.

3. Bronchoscopies should be done on these patients when they cease to raise secretion and when x-ray and physical examinations suggest patchy atelectasis.

4. Hyperventilation is the greatest benefit of bronchoscopy.

47 DEERING STREET.

REFERENCES

1. Hilding, A. C.: The Relation of Ciliary Insufficiency to Death from Asthma and Other Respiratory Diseases, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 52:5-19 (March) 1945

2. Hilding, A. C.: The Role of Ciliary Action in Production of Pulmonary Atelectasis, Vacuum in the Paranasal Sinuses, and in Otitis Media, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 52:815-833 (Dec.) 1943.

3. Vaughan, W. T., and Graham, W. R.: Death from Asthma, J. A. M. A. 119:556 (June 13) 1942.

4. Vermilye, H. N.: Aerosol Penicillin, J. A. M. A. 129:250-257 (Sept. 22) 1945.

XIII

OROPHARYNGEAL MANIFESTATIONS OF ERYTHEMA MULTIFORME

JOHN CLAIR HOWARD, JR., M.D.

KANSAS CITY, MO.

LEROY E. WIBLE, M.D.

PITTSBURGH, PA.

Many dermatologic and systemic diseases first manifest themselves by lesions in the oral cavity, nose and pharynx. A striking example of such a dermatological disease is seen in the more severe cases of erythema multiforme whose earliest symptoms are referable to the nose and throat. A review of the literature reveals that little has been written on this subject from a rhinolaryngological standpoint despite the fact that in many instances the etiology and therapeusis present a nose and throat problem.

It has been the authors' privilege to have had under their care eight cases of erythema multiforme in the last six months, all of which showed severe oral lesions. These cases were admitted to the hospital with diagnoses ranging from trench mouth to a rare blood dyscrasia. The patients arrived in a toxic and dehydrated condition, a good deal of which could have been avoided by early diagnosis and the institution of proper therapy.

The probable pathogenesis of erythema multiforme is a sensitization in the small cutaneous blood vessels of certain individuals to any one of a variety of allergenic toxic substances. It may be due to the ingestion of certain foods such as shellfish or occur as a complication of such diseases as diphtheria or measles. It is often associated with pregnancy or follows the administration of certain drugs such as the sulfonamides.[1] In many instances, even after a careful history and physical examination, it is impossible to discover any offending allergen.

It is necessary to make a distinction between the transient nonrecurring form of the disease and that type which recurs annually due to bacterial foci in the antrum and the tonsils. Such a focus of infection not only serves as a source from which micro-organisms are

disseminated into the blood stream but also and perhaps more important, it produces allergizing substances which are spread to the various tissues and sensitizes some of them.

Erythema multiforme frequently runs a very mild course characterized by vesicle formation over the body surface, the mouth, and the pharynx. However in our series, all cases were severe with an abrupt beginning followed by a stormy course.

The onset is usually characterized by a temperature ranging from 102° to 106° F. accompanied by severe malaise. The earliest lesions that can be found usually appear within the first eight hours and are seen in the oral cavity and the pharynx. They are at first bullous in appearance, later breaking down and leaving multiple necrotic ulcers over the lips, mouth and throat. The ulcers have ragged edges and are filled with a thick gray necrotic exudate. The breath is foul. Swallowing is both difficult and painful. Examination may reveal an extension of these lesions into the larynx. It is in this latter location that the prognosis is unfavorable and a tracheotomy imminent. Twenty-four to 36 hours after the onset, the bullous lesions appear on the skin and other portions of the mucous membrane. A frequent finding is the appearance of vesicles on the glans penis and the anus. In severe cases, they may be found on all mucosal surfaces of the body as well as on the skin. Postmortem findings on fatal cases have revealed lesions throughout the entire length of the gastro-intestinal tract. In many instances, there is an accompanying diarrhea.

Corneal lesions are rare but when they occur, they present a grave prognosis for vision as these ocular vesicles have a tendency to be destructive, with a resulting perforation of the cornea.[2]

Laboratory studies are disappointing for the establishment of an etiological agent. There is a moderate leucocytosis. Differential count reveals no eosinophilia.

The disease after an abrupt onset, reaches a peak usually within five days, after which the temperature becomes normal and the dermal lesions begin to disappear. There is no scar formation after healing except in the involved cornea. Pneumonitis, a not too infrequent complication, is to be feared.[3]

Several deaths have been reported in the literature. The incidence of mortality is increased when erythema multiforme occurs as a complication of a debilitating disease.

The treatment of erythema multiforme comprises local and general measures. One of the first considerations is to combat the

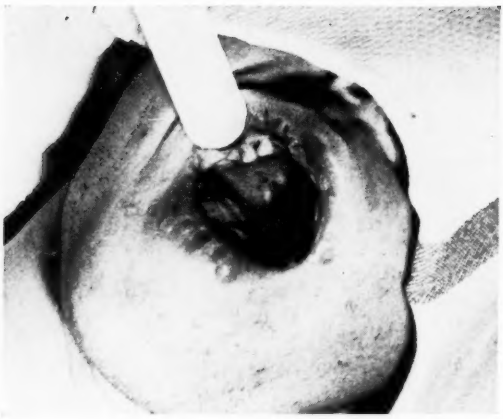


Fig. 1.—Oral lesions in erythema multiforme.

Fig. 2.—Cutaneous lesions in erythema multiforme.

dehydration and general toxic condition of the patient. This can be done by the administration of 3000 cc. of intravenous fluids daily. As long as the ulcers persist in the mouth and pharynx, it is useless to expect any fluid or nourishment to be taken by mouth. The associated diarrhea frequently adds to the excessive loss of fluids. In severe cases accompanied by anemia, blood transfusions should be given. Ten cc. of 25% calcium gluconate given intravenously every other day is helpful. Penicillin, 20,000 units every three hours, was given intramuscularly to combat secondary infection. Malted milk, vitamins and a highly nutritious diet are advisable as soon as nourishment can be taken by mouth.

Strict oral hygiene should be maintained by frequent warm saline irrigations which free the tenacious mucus which has a tendency to accumulate. Penicillin sprays to the mouth seem to be a definite help in promoting early healing. Local applications of gentian violet have been used with some success. X-ray examination of the sinuses and treatment of any local infection in the sinuses, teeth, or tonsils is definitely indicated after the subsidence of the acute involvement.

REPORT OF A CASE

R. J., aged 19, was admitted to the hospital November 10, 1945. The patient stated that he had awakened that morning with a sore throat and felt "feverish" but went to his place of employment. By noon, the sore throat was so severe that he returned home, took "some aspirin" and went to bed. His condition grew steadily worse in the afternoon. A doctor told him he had "trench mouth" and he was admitted to the hospital at five o'clock that evening with a temperature of 106° F.

Physical examination revealed a young male who appeared to be acutely ill. The lips were swollen and covered with vesicles. The oral cavity and the pharynx showed numerous thin-walled vesicles, some of which had broken down leaving ragged ulcerous craters. Breathing was somewhat difficult. There were no discernible lesions on the skin. Palatal edema was present but mirror examination revealed no encroachment of the glottis. The white cell count was 9400 and the differential showed a moderate increase in neutrophils. A diagnosis of erythema multiforme was made. The patient was placed on penicillin and oral irrigations with 0.25% pontocain. An initial dose of 40,000 units of penicillin was given to be followed by 20,000 units every three hours. A solution of 2000 cc. of 5% dextrose in physiologic sodium chloride solution was administered intravenously. The following morning the patient appeared somewhat more

toxic with a temperature of 104° F. Thin-walled vesicles began to appear on the skin and by evening the patient's body was covered with these lesions. A troublesome diarrhea developed which responded to paragoric and bismuth. Treatment was continued and after three days the patient's temperature began to subside; ten days after the onset all the lesions had disappeared.

DISCUSSION

Nose and throat pathology is undoubtedly a greater factor in the etiology of erythema multiforme than is generally realized. In this series of cases, half of them could be directly attributed to sensitization by chronic infections in the tonsils and the sinuses. It is important in the treatment of erythema multiforme to take into consideration these etiological factors. Eradication of such foci of infection after recovery is certainly good prophylaxis against repeated attacks. It behooves the otolaryngologist in the presence of any acute pharyngitis to examine the skin thoroughly for any evidence of dermatological lesions.

SUMMARY

1. Erythema multiforme is usually classed as a dermatological disease, but in many instances it is mainly an otolaryngological problem due to the severe oral and pharyngeal manifestations which may completely overshadow the underlying pathology.

2. Rhinopharyngeal foci of infection are prominent etiological factors in this disease and should be borne in mind in treatment. Eradication of these chronic foci by appropriate treatment or surgery is excellent prophylaxis against recurrent attacks.

3. A thorough examination of the skin should be routine in all cases of acute pharyngitis.

4. The etiology and treatment of a series of cases of erythema multiforme with penicillin is discussed.

1408 Professional Building
Kansas City, Mo.

516 Empire Building
Pittsburgh, Pa.

REFERENCES

1. Moss, R. E., and Long, W. E.: Toxic Eruptions Due to Phenobarbital, Arch. Derm. and Syph. 46:386 (Sept.) 1942.

2. Wheeler, J. M.: Destructive Purulent Ophthalmia Accompanying an Eruptive Fever with Stomatitis, Tr. Am. Acad. Ophth. & Otolaryn. 34:179, 1929.

3. Smith, C. A.: Unusual Case of Erythema Multiforme, Tr. Univ. Michigan Pediat. and Infect. Dis. Soc. 1929.

XIV

THE SITE OF THE FACIAL NERVE LESION IN CASES OF RAMSAY HUNT'S SYNDROME

KURT TSCHIASSNY, M.D.

CINCINNATI, OHIO

The term Ramsay Hunt's syndrome designates the occurrence of herpes zoster at the cephalic extremity, particularly at the auricle, usually associated with neural signs of the seventh or of the seventh and eighth nerves.

A brief historical review of the subject may be of some interest. Bright[1] in 1831 and Henle[1] in 1840 were the first to recognize herpes zoster as being not a disease of the skin, but rather a cutaneous manifestation of a nerve disease involving the sensory portion of the spinal nerves.

Their assumption was proved by Von Baerensprung[1] in 1861. He was able to demonstrate at autopsy in a case of herpes zoster (the first ever performed) definite inflammatory lesions at the posterior root ganglia.

Broadbent[2] in 1866 was the first to describe a case of herpetic eruption in the course of branches of the brachial plexus followed by partial paralysis in the corresponding motor nerves.

Numerous observers described cases of herpetic eruption at the cephalic extremity due to herpetic infections involving the various cranial nerve ganglia or the upper cervical spinal root ganglia.

The first description of cases of herpes zoster associated with facial palsy was given by Tryde[3] in 1872. His article was followed by numerous reports of similar observations.

From the Department of Otolaryngology, College of Medicine, University of Cincinnati.

An abstract of this paper, excluding an account of case No. 2, was given at the Postgraduate Course of the Ear, Nose and Throat Department of the College of Medicine, University of Cincinnati, May 3, 1945. Thereafter the paper in practically its present form was read before the Eye, Ear, Nose and Throat Society of Cincinnati, October 28, 1945, and at the Omaha and Council Bluffs Ophthalmological and Otolaryngological Society, January 16, 1946.

Koerner[4] in 1904 coined the term "herpes oticus" designating a syndrome which consists of the triad: blisters at the auricle, facial paralysis, and inner ear disturbances.

Ramsay Hunt[5] in 1906 emphasized, as the result of his intensive studies, the importance of the geniculate ganglion in the pathogenesis of this syndrome.

Permit me to indicate in outline the essence of Ramsay Hunt's theory. According to it, herpetic infection of the geniculate ganglion is the cause of herpes zoster appearing at a certain area of the external ear which he termed the "geniculate zone." Increased pressure of the swollen ganglion on the facial nerve produces the neurological symptoms in the area of the distribution of the seventh nerve. Occasionally associated eighth nerve signs may be explained by the simultaneous infection of the ganglia of the inner ear or by the extension of the inflammatory process from the seventh to the eighth nerve by means of a nerve filament connecting these two nerves.

Herpes appearing in an area which is supplied by the trigeminal or by cervical nerves when associated with facial or facial and eighth nerve symptoms indicate according to Hunt the simultaneous involvement of several ganglia.

In Hunt's opinion, in these cases the trigeminal or the cervical ganglion represents the main focus which is responsible for the cutaneous eruption. The inflammation of the geniculate ganglion, however, as he assumes for these cases, is not strong enough to produce the rash in the geniculate zone, and manifests itself by the aforementioned neurological signs only.

Accordingly he gives the following classification of the syndrome.

1. Herpes oticus (without neurological signs)
2. Herpes oticus with facial palsy
3 Herpes oticus with facial palsy and auditory symptoms.
4. Herpes oticus with facial palsy and Ménière's symptom complex.

With regard to the site of the rash, he differentiates:

1. Herpes oticus (geniculate herpes)
2. Herpes facialis (gasserian herpes)
3. Herpes occipito-collaris (cervical herpes)

For several years I have felt that there is some contradiction in Hunt's explanation of the pathogenesis of the facial nerve lesions in these cases. On the one hand he assumes the presence of neurological signs as a manifestation of changes in the geniculate ganglion which are strong enough to exert an effective pressure on the facial nerve. On the other hand, he assumes, to the contrary, that the occurrence of facial palsy alone is caused by a milder inflammation of the geniculate ganglion, since he assumes that these changes are not strong enough to produce the herpetic rash. Nevertheless (with the exception of a few early objections[6-8]), Hunt's concept of the syndrome has been generally accepted.

All the more interesting, therefore, is the report of Denny-Brown, Adams and Fitzgerald[9] on their histopathological findings in cases of herpes zoster. The findings at autopsy in a case of occipito-collar herpes associated with facial palsy, as reported by the authors, were: Destructive inflammatory lesions at the second cervical ganglion, patchy infiltration in the course of the facial nerve, but no changes at all in the geniculate ganglion. The authors came to the conclusion that "the evidence for geniculate ganglionitis in Ramsay Hunt's syndrome is invalid."

I suspected this several years ago when I observed a case of Ramsay Hunt's syndrome manifesting presence of taste at the anterior two-thirds of the tongue and I decided then to use the first opportunity I had to approach the problem from a clinical point of view.

The occasion presented itself last spring when I had the opportunity to observe two cases of herpes zoster associated with facial palsy, one of them an ophthalmic herpes, the other a geniculate herpes.

The question for which I sought an answer was: Are signs and symptoms presented in these cases due to the lesion of the facial nerve such as to assume the site of the lesion is at the geniculate ganglion in accordance with Ramsay Hunt's theory, or are these signs and symptoms of such a character as to localize the lesion of the facial nerve at some distance from this ganglion in contrast with Ramsay Hunt's concept?

In order to answer this question, a scheme was used which is an elaboration on the basis presented by Erb[10] in his classical work on this subject.

However, several points, which Erb obviously could not have considered in his scheme must now be recognized.

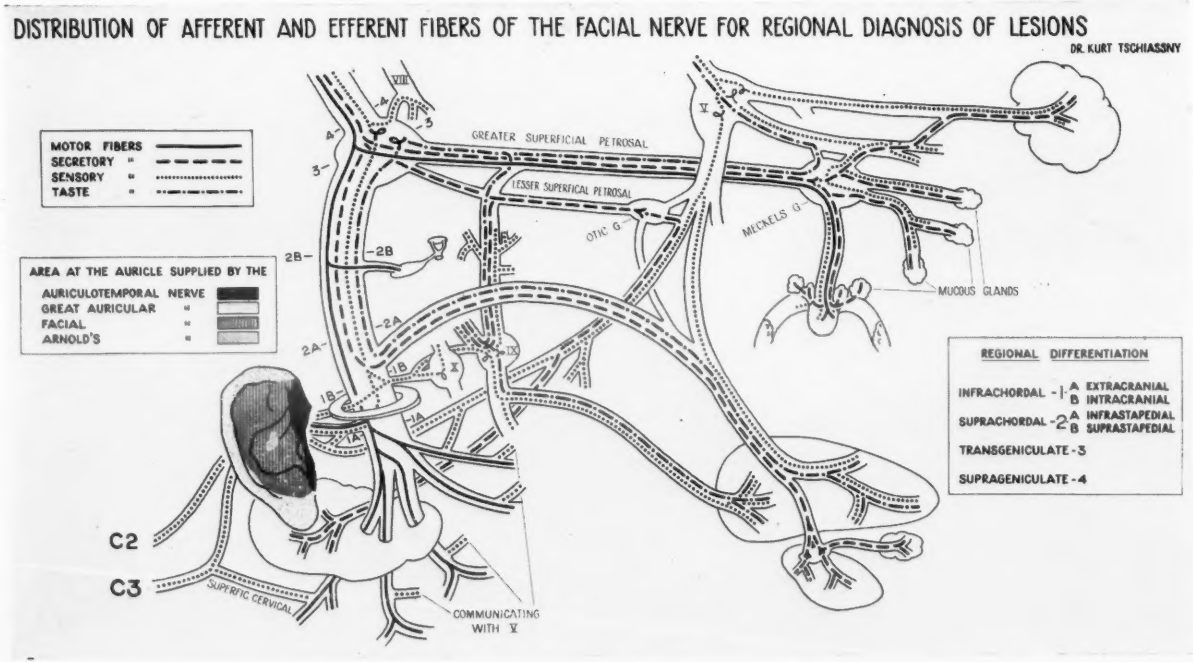

DISTRIBUTION OF AFFERENT AND EFFERENT FIBERS OF THE FACIAL NERVE FOR REGIONAL DIAGNOSIS OF LESIONS
DR. KURT TSCHIASSNY
MOTOR FIBERS
SECRETORY
SENSORY
TASTE
AREA AT THE AURICLE SUPPLIED BY THE
AURICULOTEMPORAL NERVE
GREAT AURICULAR
FACIAL
ARNOLD'S
GREATER SUPERFICIAL PETROSAL
LESSER SUPERFICIAL PETROSAL
OTIC G.
MECKELS G.
MUCOUS GLANDS
REGIONAL DIFFERENTIATION
INFRACHORDAL - 1 A EXTRACRANIAL B INTRACRANIAL
SUPRACHORDAL - 2 A INFRASTAPEDIAL B SUPRASTAPEDIAL
TRANSGENICULATE - 3
SUPRAGENICULATE - 4
C2
C3
SUPERFIC CERVICAL
COMMUNICATING WITH V

Fig. 1.

Before I present my report on these cases and the discussion of those points which are of particular interest for our problem, it might be advantageous to recall to your minds the anatomy of the facial nerve in its course through the fallopian canal, with particular reference to the function, site, and distribution of its branches. A diagram (Fig. 1) illustrates the site of branching, the course, and the anastomoses of the afferent and efferent fibers of the facial nerve.

This nerve consists of four portions which are differentiated from each other by the functionally different character of their nerve fibers. Two of them are efferent (centrifugal). They are the motor and the secretory portions. Two of them are afferent (centripetal). They are the sensory and the gustatory portions.

These fibers leave and enter the main trunk at different points. Accordingly lesions of the trunk at different levels will cause a different set of functions to be lost.

Let us now consider each portion separately with regard to the points already indicated.

1. The Motor Portion. The first group of motor fibers leaves the trunk in the greater superficial petrosal nerve. Passing through the sphenopalatine ganglion, they arrive with the palatine nerves at the soft palate for the levator veli palatini and the uvulae muscles. There exists, however, considerable controversy in the literature concerning the nerve supply of these muscles. Longet,[11] Luschka,[11] Erb,[10] Leube,[12] Chvostek,[12] Mann[13] and recently Simkins[14] (quoting Moritz), all believe that these palatine muscles are supplied by the facial nerve. Gowers,[15] Rethi,[13] Jacoby,[12] and many others are of a different opinion. There are, however, numerous observations on record of unilateral impairment of the palatinal movements in the course of facial palsy which disappeared when the facial nerve recovered.[1, 10, 13, 16-18]

Apparently, there are various ways in which these muscles obtain their nerve supply. Accordingly, intact mobility of the palate cannot be regarded as a reliable sign in the regional diagnosis of a facial nerve lesion.

The next branch is the stapedius nerve which leaves the trunk in the tympanic cavity. Contraction of the stapedius muscle draws the footplate out of the oval window. This is generally assumed to occur as a reflex which is elicited by strong auditory stimuli, particularly by loud noises or even by expectation of such a stimulus in the way of a conditioned reflex. Contraction of the stapedius muscle

was directly observed for the first time by Luescher.[19] He performed his studies on a case of traumatic perforation of the ear drum, using a magnifying glass. His findings were corroborated by later investigators.[20-23]

The stapedius reflex plainly represents a protective mechanism for the inner ear against the impact of violent sound waves. It acts for the inner ear just as the cochleo-palpebral reflex does for the eye. In both these reflexes the afferent arch is provided by the eighth nerve; the efferent arch by the seventh nerve. Contraction of the stapedius muscle produces diminution of hearing sensation. Therefore, paralysis of the stapedius muscle causes an increased hearing sensation, i.e., discomfort on loud noises, particularly in the lower frequencies. (According to Luescher's observations, however, contractions occur mainly at frequencies between 200 and 14000 d.v.)

This phenomenon has been called hyperacousis, but, as has been repeatedly observed, the term is incorrect since there is no increased acuity of the organ of hearing but only increased sensitivity for a certain type of auditory stimuli. For this reason terms such as hyperesthesia acoustica, hyperalgesia acoustica, hyperesthesia dolorosa, dysacousis, have been recommended. Obviously none of these terms is quite appropriate. In my opinion this phenomenon represents a close analogy to the ophthalmic phenomenon which is characterized by hypersensitivity for optic stimuli and generally called "photophobia." The irritating factor in the case of the eye is light (Greek phos (phot)); in the case of the ear, sound (Greek phone). Accordingly, I take the liberty to suggest the use of the word "phonophobia" for designating the symptom of hypersensitivity for auditory stimuli.

Contraction of the stapedius muscle can be brought about and made audible by voluntarily closing the eyelids with force. The strong impulse directed to the orbicularis oculi muscles spreads toward the stapedius. This contraction manifests itself as a blowing sound. The foregoing explanation was given by Lucae,[24] who first described the phenomenon.

Experimenting on this phenomenon with several doctors and students, I recognized that this noise, allegedly produced by the contraction of the stapedius muscle, is not merely a subjective sensation. It can be heard distinctly by the examiner if he will connect his ear with that of the subject by means of an auscultation tube. It can not, however, be produced by everyone and usually not equally on both sides. Based on these observations, the stapedius

muscle noise may be used occasionally as a diagnostic means. If it is present it indicates objectively the contractility of the stapedius muscle; its absence, of course, is of no significance. When, on forceful closure of the palpebral fissure, the blowing sound is audible even on the side affected by Bell's palsy, we are justified in assuming that the stapedius nerve is not involved by the lesion. This has to be located at a lower level.

The next branch leaves the trunk immediately below the stylomastoid foramen. It is the posterior auricular nerve for the rudimentary muscles of the auricle and the occipitalis muscle. Branches for the posterior belly of the digastric and stylohyoid muscles are followed by the formation of the pes anserinus. This is the digitation of the nerve trunk for the final distribution of the nerves to the muscles of expression, 55 in number, according to Moreau as quoted by Darwin.[25] All these branches are connected by communicating rami with cutaneous branches of the trigeminus, vagus, and second and third cervical nerves.

Among the muscles of expression the lacrimal portion of the orbicularis oculi muscle, so-called Horner's muscle, is of particular interest. It opens the lacrimal sac. Therefore, paralysis of Horner's muscle prevents the tears from entering the sac and causes epiphora.

2. The Secretory Portion. Fibers passing in the greater superficial petrosal nerve enter the sphenopalatine ganglion. Postganglionic fibers pass by anastomosing branches into the lacrimal branch of the ophthalmic division providing the lacrimal gland with parasympathetic fibers. Some other secretory fibers leave Meckel's ganglion for the mucus glands in the nose and palate.

A thin filament connecting the geniculate ganglion with the lesser superficial petrosal nerve and thus with the otic ganglion probably provides an additional nerve supply for the parotid gland. Wachsmuth[10] considers dryness of the mouth as a constant symptom in paralysis having its seat above the ganglion geniculi.

Finally, parasympathetic fibers are carried through the chorda tympani to the submaxillary and sublingual glands. They are relayed in the submaxillary ganglion.

3. The Sensory Portion. The sensory fibers originate in the geniculate ganglion. Their connection with the brain passes in the nervus intermedius of Wrisberg. Their peripheral distribution was the subject of intensive clinical, anatomical, pathological and embryological studies by Ramsay Hunt.

A small filament anastomosing with the eighth nerve contributes sensory fibers to the inner ear and vestigial remnants are assumed by Ramsay Hunt to pass to the middle ear, anterior two thirds of the tongue, buccal cavity, and palate. He attributes also the deep sensation of the face to the sensory portion of the seventh nerve.

Finally, he mapped out an area at the external ear, meatus, and tympanic membrane supplied by the sensory portion of the seventh nerve. This "geniculate zone" includes the tragus, antitragus, concha, part of the helix, antihelix, fossa, and a strip of the lobule. These fibers arrive at their goal mainly with Arnold's nerve and partly with the auriculotemporalis and the auricularis posterior. The rest of the external ear, meatus and ear drum is supplied anteriorly by the auriculotemporalis, posteriorly and at its posteromesial surface by the great auricular nerve and by the auricularis vagi. The last mentioned nerve anastomoses with a branch of the ninth. The nerve supply of the external ear was mapped out mainly by Cushing,[26] who examined some cases after gasserian ganglionectomy and others after lesions of the second and third cervical nerves. Thus we see that four cranial and two spinal ganglia represent areas of origin for the nerve supply of this region. It is obvious that there exists a lot of overlapping which makes the results of examination rather inaccurate.

Figure 2 shows a case of herpes zoster in the geniculate zone. Figure 3 shows a case of mandibular herpes. Figure 4 shows a case of occipitocollar herpes.

The Gustatory Portion. Taste fibers originate in the geniculate ganglion. They pass with the chorda tympani and are finally distributed to the anterior two thirds of the tongue. Some inconstant connections outside of this pathway[27] are rare exceptions and may be neglected in this study.

The central pathway, however, is still disputed. The nervus intermedius which was generally credited as the connecting link with the brain plays, if any, only a minor role in this arrangement. Many observations are on record showing complete disconnection of the facial nerve above the geniculate ganglion without loss of the sense of taste at the anterior portion of the tongue. Erb,[10] for example, referring to Stich, emphasizes that he was unable to discover in the literature of the subject or in the records of his own large experience any instance of basal paralysis of the facial nerve in which the taste sense was impaired. Wachsmuth reported a case in which both facial nerves were found to have undergone fatty degeneration in their course along the base of the skull yet taste was preserved in the anterior half of the tongue. The fibers in the greater superficial

Fig. 2.—Herpes oticus. Note the distribution of blisters at the geniculate zone.

petrosal nerve, however, were intact. Ballenger[28] in his textbook states: "Lesions central to the geniculate ganglion do not affect the sense of taste." At a meeting of the American Laryngological, Rhinological and Otological Society in 1940, Day in discussion with McCaskey[29] reported an instance of skull fracture causing complete facial paralysis with preservation of the sense of taste. He wonders how it is that in a basilar fracture involving the "nucleogeniculate" portion of the seventh nerve, the chorda tympani could escape injury. Schwartz and Weddell,[27] on the other hand, found loss of the sense of taste following injury to the greater superficial petrosal nerve without involvement of the facial nerve.

It is obvious that the central connection of the taste fibers passes mainly outside of the facial nerve.

Schiff, Erb,[10] Gowers,[15] and others assumed the fifth nerve to represent this pathway. It was, however, proved by Cushing[30] and by Mills[6] that loss of the sense of taste in the chorda tympani area of the tongue following gasserian ganglionectomy is only temporary, probably due to some neurotrophic lesions. Bing and Haymaker,[31] however, in their recent textbook still assume the taste fibers to travel with the fifth nerve toward the center.

Fig. 3.—Mandibular herpes with involvement of tongue.

Fig. 4.—Occipitocollar herpes. The blisters appear in the area of distribution of the occipitalis minor and major, the cutaneous colli, and the supraclavicularis nerves. Note a group of blisters at the lower portion of the chin which is supplied by the rami superiores of the cutaneous colli, and a few blisters at the mastoid region which is supplied by a mastoid branch of the occipitalis minor in addition to the auricularis magnus. The latter is apparently not involved in this case.

There is, of course, also another pathway to be considered. The taste fibers leave the geniculate ganglion with the greater superficial petrosal nerve and enter, by means of an anastomosis, the lesser superficial petrosal nerve. From there they pass through the tympanic plexus to Jacobson's nerve and through the petrous ganglion up to the brain stem. This is in accordance with the assumption of Krause[32] and the anatomical description given by Piersol.[33] Pette[34] reports on a case of complete hemiageusia including the anterior portion caused by a lesion of the glossopharyngeal nerve. Similar observations have been made by Dana[35] and by Cassirer according to Tigerstedt.[36]

I myself had the opportunity to examine a case after unilateral intracranial neurotomy of the ninth nerve. There was on the side of operation absence of taste over the entire half of the tongue while common sensation was present anteriorly. Unfortunately, the sense of taste had not been examined before the operation.

The exact mechanism by which gustatory impulses arrive in the brain stem is still an open question. In any case the share of Wrisberg's nerve in conduction of taste sensation is apparently negligible.

Schematic Division of the Facial Nerve. With respect to this anatomical description, the facial nerve in its peripheral course may be divided into four parts. The first and second parts may again be divided into two parts.

The four parts may be labeled as follows:

1. Infrachordal. It is the most peripheral part of the nerve. Its upper border is the branching of the chorda tympani. It consists of *A*, the extracranial, and *B*, the intracranial portions.

2. Suprachordal. It is the part above the chorda tympani as far as the geniculate ganglion. It consists of *A*, the infrastapedial, and *B*, the suprastapedial portions.

3. Transgeniculate. It is the area occupied by the geniculate ganglion including the branching of the greater superficial petrosal nerve.

4. Suprageniculate. It is the part above the branching of the greater superficial petrosal nerve.

Signs Differentiating Lesions at Different Areas. It is evident that lesions of the facial nerve located at different levels manifest

themselves by different ensembles of signs. Let us consider separately the signs presented by a lesion of each part.

Part 1A, Infrachordal-Extracranial. The paralysis usually does not involve the total peripheral distribution of the nerve. The posterior auricularis usually escapes; therefore, some movements of the auricle and of the skin behind the auricle are possible. Sensation at the geniculate zone of the auricle is not disturbed. The sense of taste and movements of the palate are normal. There is hyperlacrimation and epiphora. There are no signs of stapedius involvement.

Part 1B, Infrachordal-Intracranial. There is involvement of all branches in the peripheral distribution including the posterior auricularis. Sensory disturbances at the geniculate zone are present; otherwise the signs are as in 1*A*.

Part 2A, Suprachordal-Infrastapedial. Signs are as in the previous area with the addition of the loss of taste at the anterior two thirds of the tongue and slight diminution of salivation.

Part 2B, Suprachordal-Suprastapedial. The signs are as in the preceding area with the addition of phonophobia and necessarily the absence of stapedious muscle noise on the paralyzed side.

Part 3—Transgeniculate. The signs are as in the preceding, except that hyperlacrimation is replaced by hypolacrimation or even absence of lacrimation on the affected side. This can be proved by stimulating the nasal mucous membrane mechanically or with irritating substances ("onion test"). The uvula and the soft palate possibly deviate toward the good side. Diminution of salivation is probably more marked.

Part 4—Supragenículate. The signs are as in the preceding, but *without* loss of taste at the anterior two thirds of the tongue. (The taste fibers passing off in the greater superficial petrosal nerve escape lesion.)

Table 1. represents a simplified tabulation of the outline just described. The subdivisions are neglected for practical purposes. Taste, of course, refers to the chorda tympani region of the tongue. Mobility of the soft palate and salivation are not considered since they are regarded as inconstant. The categorical "yes" and "no", used for schematic simplicity, presupposes, of course, a complete severance of the nerve at the site of the lesion. Otherwise a qualification such as "diminished" or "disturbed" would sometimes be preferable to "no".

TABLE 1.

LOCATION OF THE LESION	PRESENCE OF TASTE	TEARS
Infrachordal	Yes	Yes
Suprachordal	No	Yes
Transgeniculate	No	No
Suprageniculate	Yes	No

It becomes evident that the regional diagnosis of peripheral facial nerve lesions is essentially based on the joint consideration of the two factors: taste and lacrimation. The four mathematical possibilities of presence or absence of these factors are correlated with four distinct parts of the facial nerve, as we have indicated above.

It may be emphasized that in facial paralysis the presence of taste at the anterior two thirds of the tongue represents entirely different topognostic signs depending on whether it is accompanied by a "wet" or by a "dry" eye. However, it may be remembered also that the presence or absence of phonophobia is of considerable significance, particularly when associated with presence of taste, the former indicating a suprageniculate, the latter an infrachordal lesion.

REPORT OF CASES

Having thus outlined this scheme for regional diagnosis of facial nerve lesions, I now present the report of the two cases as mentioned at the beginning of this paper.

Then we shall try to determine the location of the lesion in the course of the facial nerve by application of the scheme to each of the two cases in order to obtain in this way the answer to the question at issue: Where is the site of the facial nerve lesion in our cases of Ramsay Hunt's syndrome?

CASE 1.—A 58-year-old, white female was admitted to the hospital for an erythematous eruption on the left forehead and eyelids resembling erysipelas. Her temperature was 100.4° F. Other findings were normal except for the fact that the patient has been almost totally deaf and has used a hearing aid for many years. The day after admission, herpes blisters appeared at the erythematous area with some aberrant vesicles at the adjacent parts of the nose and cheek, and at the upper lip. Skin over the lateral and lower parts of the face and the external ear was normal. She was treated with penicillin, the eye was irrigated with boric acid and ophthalmic merthiolate ointment applied. In addition, irradiation was applied to the

Fig. 5.—Case 1. Ophthalmic herpes with facial paralysis.

region of the gasserian ganglion. On the seventh day after admission, facial paralysis appeared over the entire left half of the face. She could not frown, wrinkle, whistle, pucker or blow, and she showed Bell's phenomenon. She could not move the auricle on either side, but contraction of the occipitalis muscle could distinctly be felt even on the paralyzed side. Movements of the palate were normal, and the uvula was not deviated. There was increased lacrimation with epiphora. Taste was normal for all four qualities. There were no signs of involvement of the other cranial nerves. After six days the palsy began markedly to improve. Bell's phenomenon disappeared and the clusters were drying off (Fig. 5). The palsy improved rapidly within the next few days. The patient left the hospital in order to see her family doctor and did not return, so that there were no further observations.

Application of the Scheme to Case 1. Recalling the signs presented by the case reported above, there were:

1. Paralysis of the muscles of the face. There was however, reason to assume that the occipitalis muscle was not involved.

2. Normal sensation at the external ear.

3. Normal sense of taste throughout the tongue.

4. Normal salivation.

5. Signs of stapedius involvement, not to be considered because of the severe deafness.

6. Hyperlacrimation and epiphora.

7. Normal movements of the palate, uvula in the midline.

These signs particularly the presence of taste and hyperlacrimation, indicate that the lesion of the facial nerve is located below the chorda tympani, probably even extracranially, that is, in the area 1*A* of our classification.

There is no reason, therefore, to assume any involvement of the geniculate ganglion or of the facial nerve at the level of this ganglion. It is thus apparent that in this case of ophthalmic herpes the associated facial palsy cannot be regarded as due to pressure of the swollen ganglion on the facial nerve as was assumed for those cases by Ramsay Hunt.

CASE 2.—C. L., 70, colored, male, May 22, 1945. Several weeks after an upper molar tooth extraction two months previously he felt a peculiar sensation in the back of his head on the left side. Two days later he noticed some swelling and pains at the left auricle. At this same time difficulty in closing the left eye occurred. Shortly afterwards there was complete inability to move the left side of the face. The pains disappeared within a few days. The swelling at the auricle and the left hemiparalysis of the face persisted. There was no dizziness or nausea or any diminished or increased hearing sensation.

Examination on admission showed normal physical and laboratory findings except for: Left facial paralysis involving the entire motor distribution of the seventh nerve; Bell's phenomenon which was very distinctly presented; (on attempting to close the eyes, the left eyeball turned upward and outward, occasionally upward and inward) (Fig. 6). Absence of the sense of taste for all four qualities at the anterior two thirds of the tongue on the left. (Taste was present on the right side.) Movements of the tongue and the palate were normal. There was no epiphora. Experimentally, irritation of the

Fig. 6.—Case 2. Note Bell's phenomenon and absence of epiphora.

Fig. 7.—Note the appearance of a tear on the non-paralyzed side. This followed bilateral chemical stimulation.

Fig. 8.—Cutaneous changes are distinctly confined to the geniculate zone.

nasal mucous membrane by mechanical stimuli (cotton applicator) or by chemical stimuli (onion, amnonia, 40% formalin solution) caused lacrimation of the right eye only; the left does not lacrimate (Fig. 7).

The right ear was normal. The left auricle was markedly swollen. There were some crusts and some dry epidermal scales at the concha, lobule, fossa, and antihelix, including some parts of the external meatus. The ear drum was gray and glistening (Fig. 8). There were no signs or symptoms indicating involvement of the vestibular apparatus.

An audiogram (Fig. 9), repeated several times, presents for both ears a curve typical for the age of the patient.

Tones of higher intensities were felt to be louder on the left than on the right, particularly those in the range between 225 and 1012 d.v. Simultaneously a peculiar tickling sensation occurred. This sensation was felt mainly when intensities between 50 and 85 db. above his hearing threshold were applied. It was not felt at intensities below, and less distinctly at intensities above this range. (These findings present to some extent a similarity with findings reported by Perlman[23] in a case of facial paralysis.) When an intensity of 100

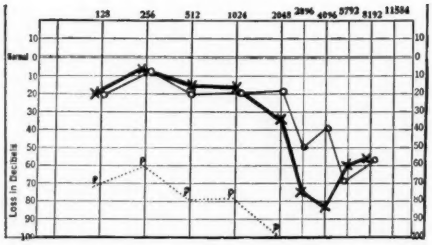


Fig. 9—Case 2. Audiogram made May 26, 1945. Left ear X-X-X, right ear O-O-O. The dotted line (P . . .P . . .) indicates the appearance of phonophobia. Note the almost complete parallelism with the curve of threshold.

db. for 256 or 512 d.v. was abruptly applied at the left ear, a disagreeable sensation was produced which caused reflexly a sudden fugitive movement of the head. This did not occur when the same stimulus was applied at the right ear. Stapedius muscle noise was distinctly audible in the right ear, not audible in the left. In tests for common sensation, he differentiated between sharp and blunt over the whole auricle except at the concha where mistakes repeatedly occurred.

The patient was treated with vitamin B complex, nicotinamide, infra-red and x-rays and application of electric currents. The cutaneous lesion improved rapidly and disappeared completely after two weeks. There was however no improvement in the neurological lesions except for partial disappearance of the phonophobia and reappearance of lacrimation, with epiphora after several weeks. There were otherwise no changes observed for the next five months. At this time a new pathological sign appeared which was first observed by the patient himself. He noticed the appearance of grayish-green, bad smelling crusts in the left nostril. He had considerable difficulty in getting those crusts out of the nose. Sometimes these attempts were followed by nose bleeds. These crusts were in the left nostril only.

Examination of the nose revealed a condition which can be best described as a unilateral ozena. There was marked atrophy of the turbinates, the left side being considerably wider than the right. The middle meatus was filled with a fetid crust of inspissated secretion. X-ray findings of the sinuses were normal. The patient was advised to use regular irrigations and one per cent menthol in cod liver oil intranasally.

At this time I presented the patient to the staff. This condition has not markedly changed since it first appeared.

Comment: The development of an ozena on the side of the facial nerve lesion is obviously due to the paralysis of the secretory fibers for the nasal mucous glands passing in the greater superficial petrosal nerve to Meckel's ganglion. This is apparently a rather rare occurrence. I was not able to find in the literature a report of ozena occurring in the course of facial nerve paralysis although a few cases of unilateral ozena due to lesions of the sphenopalatine ganglion are on record.

The reappearance of lacrimation in this case is hard to understand. The development of the ozena makes the assumption of an isolated recovery of the parasympathetic portion of the facial nerve rather unlikely. Probably some other nerve connection passing from the otic or ciliary ganglion took over the function.

The distinct diminution of phonophobia is probably due to some kind of central adaptation.

This case, of course, will be the subject of further observation and bacteriological and histological examination. The final result will be reported later.

Application of the Scheme to Case 2. Summarizing the signs presented by this case of herpes oticus (geniculate) associated with facial palsy, there were:

1. Complete facial hemiparalysis.

2. Disturbance of common sensation at the concha.

3. Loss of taste at the anterior two thirds of the tongue on the paralyzed side.

4. Distinct phonophobia.

5. No epiphora but unilateral loss of lacrimation lasting for several weeks.

6. Neurotrophic changes in the nose on the side of the paralysis manifesting themselves as unilateral ozena.

These signs indicate that the lesion of the facial nerve in this case of herpes oticus is located at the area where both the taste fibers and the secretory fibers are in a position to be involved. This is area 3 of our classification, the transgeniculate. In this case, therefore, there is good reason to assume an involvement of the geniculate gang-

lion. Thus it is very well to assume that pressure due to the inflammatory swelling of the geniculate ganglion on the facial nerve causes damage to this nerve conforming completely with the theory of Ramsay Hunt.

Application of the Scheme to Cases in the Literature. It is of interest to review in connection with the presented scheme cases of herpes zoster reported by various authors.

Unfortunately, comparatively few of these clinical reports are exact enough to be used for the purpose of this study. There were reviewed altogether only 22 cases reported by the following authors: Darabseth,[37] Ebstein,[1] Fraser,[38] Hammerschlag,[16] Haymann,[17] Hunt,[18] Kaufmann,[16] Spillane,[39] Struebing,[40] Tryde[3] and Voigt.[41]

Table 2 presents a collection of these cases with the site of the cutaneous eruption and the site of the nerve lesions in each group reclassified according to symptomatology as outlined above.

TABLE 2.

SITE OF HERPETIC RASH	NUMBER OF CASES	SITE OF FACIAL NERVE LESION			
		INFRA-CHORDAL	SUPRA-CHORDAL	TRANSGEN-ICULATE	SUPRAGEN-ICULATE
Occipitocollar	10	5	5	--	--
Facial	5	3	2	--	--
Geniculate	7	--	2	5	--

Comment: This review shows that out of seven cases of geniculate herpes, five presented evidence of involvement of the facial nerve at the level of the geniculate ganglion.

In 15 cases with the cutaneous eruption in the gasserian or cervical zone none presented signs indicating a lesion of the facial nerve at the level of the geniculate ganglion. It may be of interest to note that in some of these cases the sense of taste was undisturbed at the beginning of the facial palsy but was lost later on in the course of the disease.[18, 39]

These observations conform with the statement of Fenton,[42] Mills, et al., that herpes zoster has to be considered as an ascending or descending infective process. Numerous anastomoses of the branches of the seventh nerve with the cutaneous peripheral distributions of the trigeminal, of Arnold's nerve, and of the spinal nerves probably provide a portal of entry into the seventh (Fig. 1).

CONCLUSIONS

Analysis of the reported case of ophthalmic herpes and review of 15 cases in the literature indicate that the lesion of the facial nerve, when its palsy is associated with facial or occipitocollar herpes, can be located as a rule at a level below the geniculate ganglion but the ganglion itself is not involved. These findings conform to some extent to the autopsy findings of Denny-Brown, Adams and Fitzgerald, but do not conform with Ramsay Hunt's theory.

Cases, however, with the herpetic eruption at an area of the external ear which is, according to Hunt, supplied by the sensory portion of the seventh nerve (such as Case 2 reported here and seven cases reviewed in the literature) present, in the overwhelming majority, signs indicating the site of the lesion at the level of the geniculate ganglion. These findings do conform to Ramsay Hunt's theory.

Therefore, it becomes evident that facial paralysis, associated with gasserian or cervical herpes, is essentially different in its pathological and clinical aspect from facial paralysis associated with herpes oticus.

In the former group, evidently, the geniculate ganglion is not involved at all, contrary to Ramsay Hunt's theory; in the latter group, as a rule, the geniculate ganglion seems to be involved, conforming with Ramsay Hunt's theory.

It may, therefore, be even justifiable to restrict the term "Ramsay Hunt's syndrome" exclusively to instances of the herpes oticus syndrome, while cases of herpes facialis or herpes occipitocollaris syndrome should be classified separately.

SUMMARY

A brief historical review of herpes zoster, with particular reference to herpes zoster oticus, is presented.

The pathological findings of Denny-Brown and co-workers contradicting Ramsay Hunt's geniculate ganglion theory are recalled.

The problem is reexamined by the present author from a clinical point of view in connection with two cases of herpes zoster of the cephalic extremity associated with facial palsy. The question turns on determining the site of the lesion in the facial nerve in these cases. For this purpose the anatomy of the afferent and efferent portions of the facial nerve and their branches and distributions are described. Topognostic signs for lesions of the facial nerve in its course through

the fallopian canal are described, analyzed, and presented in a simplified tabular scheme.

The stapedius muscle reflex is discussed.

The possibility of a new objective method for diagnosis of stapedius muscle activity is suggested.

The term "phonophobia" for designating hypersensitivity to loud noises is recommended.

The two presented cases are analyzed in accordance with this scheme.

In the first reported case of ophthalmic herpes the lesion has to be located at a level below the chorda tympani. Therefore, there is no reason to assume an involvement of the geniculate ganglion, which is in contrast to Ramsay Hunt's theory.

In the second reported case of geniculate herpes the lesion, according to the signs, has to be located at the geniculate ganglion. This case tends to confirm Ramsay Hunt's theory. Attention is called to the symptom of phonophobia in this case.

Relevant audiometric records are given.

The unusual observation of unilateral ozena developing in the course of facial nerve paralysis in this case is described.

Twenty-two cases in the literature are reviewed with regard to this scheme. This study shows that the lesion of the facial nerve when its palsy is associated with facial or with occipitocollar herpes in no instance can be located at the geniculate ganglion, while it must be located at this ganglion almost regularly when associated with herpetic eruption at the "geniculate zone" of the external ear.

It is suggested to restrict the term "Ramsay Hunt's syndrome" exclusively to instances presenting herpes zoster in the "geniculate zone" of the external ear.

3507 Burnet Avenue.

REFERENCES

1. Ebstein, W.: Herpes zoster und Facialislaehmung, Virchow's Arch. 139: 505, 1895.

2. Head, H., and Campbell, A. W.: The Pathology of Herpes Zoster and Its Bearing on Sensory Localization, Brain 23:353, 1900.

3. Tryde, C.: Bemerkungen zur Facialisparese, Schmidt's Jahrb. 2:272, 1872.

4. Koerner, O.: Ueber den Herpes Zoster Oticus (Herpes an der Ohrmuschel mit Laehmung des nervus acusticus und des nervus facialis), Muench. med. Woch., page 6, 1904.

5. Hunt, J. R.: Inflammation of the Geniculate Ganglion, A New Syndrome and Its Complications, Trans. Amer. Neur. Assn., p. 184, 1906.

6. Mills, C. K.: The Sensory Function Attributed to the Seventh, J. Nerv. & Ment. Dis. 37:273, 355, 1910.

7. Kidd, L. J.: The Alleged Sensory Cutaneous Zone of the Facial Nerve in Man, Rev. Neuro. & Psych. 12:393, 1914.

8. Orbison, T. J.: Herpes of the Membrana Tympani due to Zosterial Affection of the Petrosal Ganglion, J. Nerv. & Ment. Dis. 35:8, 1908.

9. Denny-Brown, D., Adams, R. D., and Fitzgerald, P.: Pathologic Features of Herpes Zoster, Note on "Geniculate Herpes", Arch. Neur. & Psych. 57:216 (March) 1944.

10. Erb, W. H.: Ziemssen Cyclopedia of Practice of Medicine, Vol. 2, page 490, 1876.

11. von Luschka, H.: Der Schlundkopf der Menschen, Tuebingen, H. Laupp, 1868.

12. Jacoby, G. W.: Peripheral Facial Diplegia, J. Nerv. & Ment. Dis. 73, 1907.

13. Mann, M.: Ueber Gaumenlaehmung, Ztschr. f. Ohrenh. 47:1-39, 1904.

14. Simkins, C. S.: Functional Anatomy of the Eustachian Tube, Arch. Otolaryng. 38:476, 1943.

15. Gowers, W. R.: A Manual of Diseases of the Nerve System, 2:241, 1900.

16. Hammerschlag, V.: Die Rheumatischen Affectionen des Gehoernerven-apparates, Arch. f. Ohrenh., p. 16, 1901.

17. Haymann, L.: Ueber Zostererkrankungen im Ohrgebiete, Ztschr. f. Hals-Nasen- u. Ohrenh. 1:396, 1922.

18. Hunt, J. R.: Geniculate, Auditory, Glossopharyngeal and Vagal Ganglia, Arch. Int. Med. 5:631 (June) 1910.

19. Luescher, E.: Die Function des Musculus Stapedius beim Menschen, Ztschr. f. Hals- Nasen- u. Ohrenh. 23:105, 1939.

20. Kobrak, H., Lindsay, J. R., and Perlman, H. B.: Value of the Reflex Contraction of the Muscles of the Middle Ear as an Indicator of Hearing, Arch. Otolaryng. 21:663 (June) 1935.

21. Lindsay, J. R., Kobrak, H., and Perlman, H. B.: Relation of the Stapedius Reflex to Hearing Sensation in Men., Arch. Otolaryng. 23:671 (June) 1936.

22. Potter, A. B.: Function of the Stapedius Muscle, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 45:638-643, 1936.

23. Perlman, H. B.: Hyperacousis, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 47:947, 1938.

24. Lucae, A.: Die Accomodation und die Accomodation-stoerungen des Ohres, Berl. klin. Wchnschr., 1874.

25. Darwin, Charles: The Expression of the Emotion in Man and Animal, New York, D. Appleton-Century Co., 1929.

26. Cushing, H.: The Sensory Distribution of the Fifth Cranial Nerve, Bull. Johns Hopkins Hosp. 15:213, 1904.

27. Schwartz, H. G., and Weddell, G.: Observations on the Pathways Transmitting the Sensation of Taste, Brain 61:99, 1938.

28. Ballenger, W. L.: Diseases of the Nose, Throat, and Ear, Philadelphia, Lea and Febiger, 1938.

29. McCaskey, C. H.: Etiology of Seventh Nerve Paralysis, Tr. Am. Laryng. Rhin. & Otol. Soc., p. 294, 1940.

30. Cushing, H.: The Taste Fibers and Their Independence of the Nervus Trigeminus, Bull. Johns Hopkins Hosp. 14:71, 1903.

31. Bing, R., and Haymaker, W.: Regional Diagnosis in Lesions of the Brain and Spinal Cord, 11th ed., St. Louis, C. V. Mosby Co., 1940. p. 155,

32. Dixon, F.: Further Note on the Course of the Taste Fibers, Edinbourgh M. J., p. 628, 1897.

33. Piersol's Human Anatomy, edited by Kuber, G. C., 9th ed. rev., Philadelphia, J. B. Lippincott, 1930, p. 1247.

34. Pette, H.: Zur Symptomatologie und Differential Diagnose bei Kleinhirnbrueckenwinkeltumoren, Arch. f. Psych. und Nervenkr. 64:98-132, 1922.

35. Dana, C. L.: Discussion to Taylor, C. G., J. Nerv. & Ment. Dis. 37:323, 1910.

36. Tigerstedt, R.: Lehrbuch der Physiologie des Menschen, Leipzig, S. Hirzel, 1908, p. 126.

37. Darabseth, N. B.: Bell's Palsy following Herpes Zoster, Recovery, Lancet, p. 1129, 1894.

38. Fraser, E. H.: A Case of Facial Paralysis Associated with Herpes Zoster, Lancet, p. 18, 1904.

39. Spillane, J. D.: Bell's Palsy and Herpes Zoster, Brit. M. J. 1:236, 1941.

40. Struebing: Herpes Zoster und Laehmung Motorischer Nerven, Deutsch. Arch. f. Klin. Med. 47:577, 1885.

41. Voigt, E.: Ueber Complication von Herpes Zoster Occipitocollaris mit schwerer gleichzeitiger Facialisparalyse, St. Petersburger med. Wchnschr. 45:449, 1884.

42. Fenton, R.: Diagnostic Features concerning Herpes Zoster Oticus, J. A. M. A., p. 468, 1934.

Clinical Notes

XV

FOLLICULAR ODONTOMA OF THE MAXILLARY SINUS

SAM E. ROBERTS, M.D.

FRANK S. FORMAN, M.D.

KANSAS CITY, MO.

Follicular odontomata are tumors arising from tissue having to do with tooth development. This type of tumor is so rare that we thought this case should be placed in the literature. As near as we can determine from the literature there are less than 50 cases of odontoma in the antrum reported. Geschikter and Copeland[1] in a survey of 323 tumors of the jaw found only four cases of odontoma. Perthes[2] states that while hard odontomata are rare, they occur more frequently than soft odontomata.

Nolan and Kennedy[3] give an excellent review of the literature including the classification of the British Dental Association, which is as follows:

A. Epithelial odontoma:
   - (a) Adamantinoma
   - (b) Dentigerous cyst
   - (c) Dental cyst

B. Composite odontoma:
   - (a) Complex composite odontoma
     Compound composite odontoma
   - (b) Germinated composite odontoma
     Gestant composite odontoma
     Enamel nodules
   - (c) Dilated composite odontoma

From the Department of Otolaryngology, University of Kansas School of Medicine.

C. Connective tissue odontoma:
   (a) Fibrous odontoma
   (b) Cementoma

According to Bernier[1] "They may be further classified, according to Bland and Sutton (revised 1914), in relation to the particular tissue in their development, as follows:

(a) Epithelial odontoma (origin within ectodermal tissue)

(b) Composite odontoma (origin primarily within ectodermal tissue and secondarily within mesodermal tissue)

(c) Connective tissue odontoma (origin within mesodermal tissue)."

Follicular odontomata are classified as "ectodermal" in origin and may be either simple or compound, depending upon the number of teeth within the cystic cavity.

The term "composite odontoma" is used to include all odontomata in which the abnormal development takes place primarily in the dental epithelium and secondarily in the dental papilla and follicle.

Complex composite odontomata consist of a conglomerate mass of tooth structures, enamel, dentin and cementum, with the tooth structure shown in microscopic sections. Compound odontomata are encapsulated tumors containing numerous teeth or denticles consisting of enamel, dentin or cementum. The many small enamel organs that are formed instead of a single normal enamel organ may be separate in the cavity or fused. They develop early in life and are usually associated with unerupted teeth.

These tumors are new growths. All of the contributors to the pathogenesis consider the dental follicle a factor in the formation of these cysts except Harris,[5] who believes that any disease process affecting the enamel membrane may prevent eruption, the internal epithelium being the source of cystic fluid. Fisher[6] considers inflammatory disturbances or tissue irritation to inaugurate a proliferative change. Malassez[7] believes there is a hypertrophy of the epithelial rudiments of the enamel organs. Dorrance[8] considers the cyst to arise from the enamel sac of an abnormal tooth.

However it is safe to say that it is during the earlier stages of development of the tooth germ that odontoma formation is first noted. An understanding of the periodic phases of this development

is necessary to follow the morbid changes as they occur. About the fortieth day of intra-uterine life the earliest stage of the tooth germ is noted. There is a proliferative change resulting in a slight thickening of the stomodeal ectoderm over the alveolar process.

Hertzler,[9] for the purpose of clarity in treatment, looks on follicular odontoma as analogous to dermoids. If all the lining is destroyed either by surgical removal or chemical means, the cyst remains cured. If a portion of the lining escapes destruction a renewed cyst or permanent fistula results. For this reason curettement often results in an incomplete cure. Cavina[10] discusses three types of surgical procedures that may be carried out: first, total exploration on the cyst, curettage of the mucosa of the maxillary sinus and a nasal counter opening; second, transformation of the cyst into an accessory nasal cavity; third, wide communication of the cyst with the antrum demolishing the cystic sinual septum and making a nasal counter opening. "When possible it is always best to remove the cyst. The second type of operation is used when the cyst is not greatly developed and the cyst is in the anterior and interior part of the antrum. The third operation is used only in those rare cases in which it is not practical or opportune to expirate the cyst and curet the entire mucosa and there is a reason to believe that by changed conditions the maxillary sinus has not free communication with the nose by its orifice."[10]

REPORT OF A CASE

A boy, aged 12 years, was first seen March 4, 1945, with the following history: his mother noticed swelling of the right cheek for the past three months. X-ray films had been taken at another hospital, January 12, 1945. The true picture was evidently not revealed at that time and the family was advised to use heat (hot packs). X-rays taken March 5, 1945 revealed two displaced teeth, one near the orbital floor and the other apparently lying in the inferior meatus. The interior wall was largely destroyed and these teeth were removed along with the tumor mass. The third tooth found at operation was not visualized by x-ray.

A soft mass could be palpated at the right central incisor extending back to the posterior molar region. The nose was completely obstructed on that side.

A Caldwell-Luc technique was carried out. Careful incision just through the mucosa was made so the cyst would not be ruptured. The outer wall of the maxillary sinus had been completely destroyed by erosion. With blunt dissection and gauze the tumor mass was removed and was not ruptured until it was loosened from the floor of the max-

illary. The cavity was very thoroughly curetted. All teeth removed in the cyst were well developed.

So far the boy has made an uneventful recovery but the parents have been warned of a malposed tooth on the opposite side which may be another odontoma. Only one tooth in the alveolar ridge was removed at the time of operation although two more were loosened but seem to be tight at this time (ten months later).

1110 Professional Bldg.
Kansas City, Mo.

REFERENCES

1. Geschikter, C. F., and Copeland, M. M.: Tumors of Bone, Rev. Ed., New York, Amer. Jour. Cancer, 1936.

2. Perthes, G.: Die Vorlotzungen und Krankheiten der Kiefer, Deutsche chir. 33A:359, 1907.

3. Nolan, L. E., and Kennedy, C. L.: Review of odontoma with Case Report, J. A. D. A. 29:389-395 (March) 1942.

4. Bernier, J. L.: Follicular Odontomata, Mil. Surgeon 87:49, 1940.

5. Harris, J. H.: Pathology of Tooth Containing Cysts and Its Bearing on Eruption, Brit. Dental J. 35:1, 1934.

6. Fisher, Guido: Experimental Researches on the Development of Follicular Cysts, Dental Cosmos. 53:1218, 1911.

7. Malassez, L.: Sur le role des dibris epitheliaux paradentairis, Arch. de physiol., norm et path. 17:379, 1885.

8. Dorrance, G. M.: Etiology, Pathology and Treatment of Cysts of the Jaw, J. A. M. A. 77:1883 (Dec. 10) 1921.

9. Hertzler, A. E.: Surgical Pathology of the Mouth and Jaw, Philadelphia, J. B. Lippincott Co., 1938, p. 163-166.

10. Cavina, Caesar: Three Operative Procedures and Treatment of Dental Radicular Cysts of Maxillary Bone, M. J. & Rec. 129:488-493 (May) 1929.

XVI

A CASE OF OSTEOMYELITIS OF THE FRONTAL BONE EXTENDING FROM THE FRONTAL SINUS, COMPLICATED BY MENINGITIS, RECOVERY

FREDERICK T. MUNSON, M.D.

AND

MORRIS ZUKERMAN, M.D.

DETROIT, MICH.

Osteomyelitis of the facial bones occurs either after surgical operations as a result of an extension of the infection from diseased sinuses or possibly following injury. Once the infection of the bone has set in, there is a tendency for the inflammatory process to spread to the brain and the meninges. Osteomyelitis of the frontal bone has been attributed to various organisms including staphylococcus aureus and anaerobic nonhemolytic streptococci. These germs usually reach the bone through an infected frontal sinus.

Operative procedures and the use of sulfonamides has decreased the mortality rate somewhat, but in severe cases the latter may be as high as 80 per cent. With the advent of the use of penicillin, a much better prognosis of a case is possible. Kirby and Hepp[1] have reported five cases of osteomyelitis of the facial bones treated with penicillin and surgery with recovery in each case.

Williams and Nichols[2] reported two cases of spreading osteomyelitis of the frontal bone treated with penicillin and surgery with recovery. It seems necessary to stress the fact that surgery must be combined with penicillin in most cases if a cure is to be obtained.

It has been recommended that the surgical procedure be postponed until the patient has had adequate penicillin and until sequestra have formed.[1] Others have felt that where there is a fulminating sinusitis, frequently caused by the anaerobic streptococcus, it is necessary to create openings into the floor of the frontal sinus and irrigate with hydrogen-peroxide to establish aerobic conditions in the sinus. Penicillin is to be used in conjunction with this form of treatment.[3]

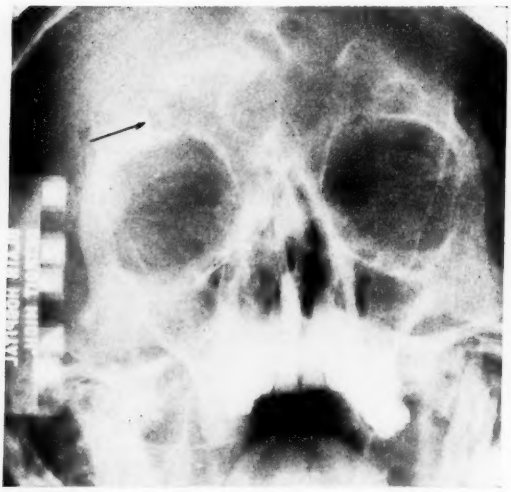


Fig. 1.—Roentgenogram showing osteomyelitis of the frontal bone with sequestra, indicated by arrow.

Our patient, a 48-year-old Italian male presented himself at the clinic on June 30, 1945. The present illness began on June 15, 1945, when the patient complained of a severe aching pain in the right side of his face. This persisted until he entered another hospital on June 18, 1945. Associated with this pain was a slight, purulent nasal discharge, and the passage of bright red blood from the nose. There was no radiation of the pain. He had had an upper respiratory infection for several days before the acute onset of pain described. There was a history of chronic sinusitis for "several" years.

Prior to entering our hospital, he was treated with penicillin, 40,000 units intravenously followed by 30,000 units intramuscularly every two hours day and night. This had been started on June 18. Under this therapy the nasal discharge gradually subsided and the patient's general condition improved. He was released from the hospital on June 23.

When the patient was first seen by us he stated that on June 27, four days after leaving the first hospital, he noticed a painful swelling over the right eye and was advised to have medical attention at once. The patient was immediately hospitalized.

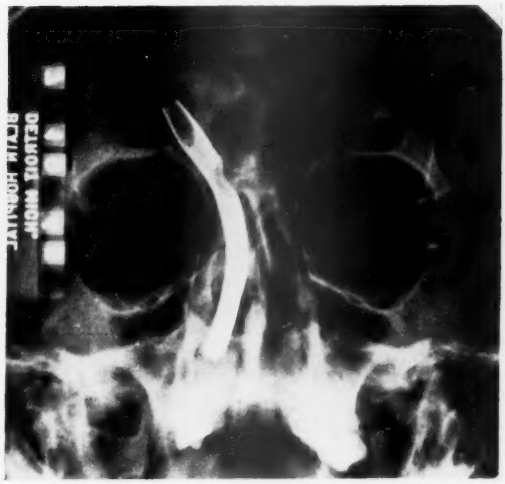


Fig. 2.—Roentgenogram showing sinus after removal of diseased bone (arrow), and tube in place.

Physical Examination. The physical examination upon entrance to the hospital showed the following: temperature 99.6; pulse rate 80; respiration 23. His general appearance was of an acutely ill, markedly undernourished person. His skin was warm and dry. The teeth showed moderate caries. Eyes: His vision was 20/20, the pupils were round and equal, and reacted to light and accommodation; the fundi showed no areas of hemorrhage or degeneration. The maxillary sinuses were dark on transillumination. There was a large, red, swollen area just over the right eye (the area about the brow, which was extremely tender to touch). The eye was practically closed with swelling and a doughy, puffy area was noted over the frontal bone extending upward to the hairline.

In the neck there was no stiffness, palpable masses or redness along the cervical veins. The examination of the lungs revealed hyperresonance on percussion. The heart was not enlarged; no murmurs but occasional extrasystoles were heard.

Diagnosis. The following diagnoses were made:

1. Right frontal sinusitis with osteomyelitis of the frontal bone.

2. Undernutrition.
3. Pulmonary emphysema.

Laboratory Work. Laboratory work on admission to our hospital was as follows:

Hb.		92%
RBC		4,584,000
WBC		12,500
Differential Count:	Neutrophiles	75%
	Non-filamented	9%
	Lymphocytes	16%
Kahn and Kline Tests		Negative
Urine		Negative

X-rays. The x-ray report on the sinuses from the hospital first consulted by the patient was as follows: "There is a diffuse clouding of both frontal sinuses which leads us to presume that there probably has been a communication between the two sinuses. Destruction of the anterior wall of the smaller right frontal sinus is evident and we believe that there is a complicating osteomyelitis. The maxillary sinuses are both clouded."

On June 30 an x-ray of the sinuses was interpreted as follows: There are fairly large frontal sinuses bilaterally but there is marked asymmetry of shape and size. The right half of the right frontal shows irregular walls and some increase in density at the margins. There is definite clouding of all the right ethmoids and the right maxillary sinus while the left maxillary also shows some involvement.

Conclusion. Pansinusitis involving all except the left ethmoids. On the right frontal an irregularity of the wall suggests osteitis and sinusitis with possible osteomyelitis and sequestra. On June 30 the patient was taken to the operating room where a trephine opening was made in the anterior wall of the right frontal sinus just above the inner angle of the right eye. A large amount of thick, creamy pus came from this opening.

As soon as the patient was taken to his room penicillin was started by continuous intramuscular drip; 2,000,000 units were given. On July 6, about one week later, the patient was again taken to the operating room where an external exploratory operation of the right frontal sinus and frontal bone was performed. Findings at the time of the operation were as follows: There was a sequestrum just above the brow about half way between the centerline and the outer angle

lying in the anterior wall of the frontal sinus. This was removed and the other diseased bone adjacent to it was cut away with a rongeur. The frontal sinus was found filled with polypoid material and with pus.

Almost the entire anterior wall of the right frontal sinus was cut away, the contents were curetted out and the left area was explored with suction. There was no bony septum encountered and the contents of the relatively large left sinus were evacuated. The right nasofrontal duct was enlarged and sufficient bone was curetted away to place a large catheter through into the nose. This was allowed to remain in place. The skin and periosteum were sutured and closed completely and dressings applied. After the operation penicillin was again started by continuous intramuscular drip, 2,000,000 units again being given.

On July 10, four days later, the patient began to complain of marked soreness of his thighs as a result of the continuous drip, so that the amount was decreased to 360,000 units per day, given intramuscularly every two hours. On July 13 the temperature returned to normal and remained so until five days later when there was a spike of temperature, accompanied by a rather severe leftsided headache. Penicillin had been discontinued on July 15. On July 19 the patient was given sulfadiazine, grains 15, four times daily. On July 22 penicillin was resumed, 30,000 units being given every two hours intramuscularly, and the sulfadiazine was increased to 90 grains daily.

The following day the patient began to complain of more severe headaches and stiffness of the neck. A lumbar puncture was performed immediately which confirmed the clinical impression of meningitis. On July 24 sulfadiazine, grains 30, and soda-bicarbonate, grains 15, were given five times daily. Penicillin was also started, 2,000,000 units being given daily by continuous intramuscular drip. The clinical improvement continued on this regime and on August 2 the penicillin was discontinued, but the sulfadiazine was continued until his discharge from the hospital August 11, 1945, as cured. There was, however, a residual numbness of the dorsum of the right foot. When seen in the out-patient department on August 23 and September 14, the patient stated that he continued to feel well except for the numbness of his right foot.

An x-ray of the sinuses on November 27 showed hyperplastic sinusitis. There was also an irregularity of the right frontal sinus and considerable thickening of the wall denoting a condensing osteitis, a result of the old osteomyelitis.

SUMMARY AND CONCLUSION

(1) A review of the literature pertaining to osteomyelitis of the frontal sinuses is presented.

(2) The mortality rate has fallen sharply by combining surgery, sulfonamides and penicillin.

(3) A case report has been presented of complete recovery from acute osteomyelitis of the frontal bone, arising from frontal sinusitis and complicated by meningitis.

19962 Renfrew St.

REFERENCES

1. Kirby, William, and Hepp, V. E.: Treatment of Osteomyelitis of the Facial Bones with Penicillin, J. A. M. A. 125:1019-1022 (Aug. 12) 1944.

2. Williams, H. L., and Nichols, D. R.: Spreading Osteomyelitis of the Frontal Bones Treated with Penicillin, Proc. Staff Meetings, Mayo Clinic 18:467 (Dec. 1) 1943.

3. Williams, H. L.: Chemotherapy in Otolaryngology, Wisconsin Med. J., May, 1945.

XVII

BILATERAL GRANULOMA OF THE LARYNX FOLLOWING INTRATRACHEAL ANESTHESIA

HAROLD LESLIE KEARNEY, M.D.

NEW ORLEANS, LA.

Intratracheal anesthesia has been used now for many years and in many thousands of cases by hundreds of anesthetists. It has been a boon to both surgeon and patient and many surgical procedures would be impossible without it, particularly in thoracic surgery. Its benefits greatly outweigh its few undesirable sequelae. One of these sequelae is polypoid granuloma of the larynx. Smiley[4] reported a case in 1940 and in a review of the literature to that time found only three other cases of this complication reported. My review of the literature since 1940 discloses no additional reports. The following report therefore makes a total of five cases in the literature.

Mrs. A. E., aged 49 years, had a cyst of the left upper alveolus, which was operated on by way of the radical antrum approach on June 24, 1944, under intratracheal ethylene ether anesthesia through a Magill tube. Preoperative examination showed a normal larynx. Nasal intubation was done by the anesthetist, who guided the tube into the larynx with moderate difficulty under direct vision with the direct laryngoscope. Anesthesia was administered for one hour and 25 minutes, the operation being longer than was expected, because of bleeding. On recovery from the anesthetic the patient complained of pain in the throat which subsided in about a week.

On July 28 she again had pain in the throat in the region of the larynx on the left side and was somewhat hoarse. Mirror examination of the larynx showed a granuloma on the posterior third of each cord in the region of the vocal process. On August 9 the granulomata were definitely pedunculated. Operation was advised but was postponed on the advice of her internist who found a slight anemia and a moderate neutropenia. On August 22 the respiration had become slightly noisy and appointment for operation was made. However at 3 A. M., August 23, she became dyspneic and was admitted to the hospital as an emergency and the granulomata removed under

Presented before the Southern Section of the American Laryngological, Rhinological and Otological Society, Richmond, Va., January 7, 1946.

ether anesthesia. The operation was simple. The larynx was exposed by the anterior commissure laryngoscope and each granuloma was removed by one grasp of the angular vocal nodule forceps on its pedicle. Following the operation the larynx immediately appeared normal. There were no further laryngeal symptoms and inspection of the larynx 16 months later still showed a normal larynx. The report of the pathologist was "granulation tissue polypi of the larynx."

A common feature of the cases reported by Clausen[1] and Cohen[2] is that the lesion arose in the posterior portion of the larynx. The case reported by Gould[3] and the one by Smiley[4] were designated as subglottic lesions and were not more definitely localized. In the case herein reported the lesions arose from the region of the vocal process of each vocal cord. This suggests to me that the lesion may generally originate in a traumatic ulcer of the cord exposing the superficial cartilaginous vocal process of the arytenoid. A granuloma forms and continues to grow and meanwhile the ulcer heals slowly from the periphery. This produces a smaller and smaller base for the lesion and gradually a pedicle is formed. In the case reported by Clausen,[1] this healing process may have progressed to the point of amputation of the pedicle, as the patient coughed up the pea-sized tumor about six months after her intratracheal anesthetic. All the other reported cases were cured by surgical removal of the tumor. However in two cases, those of Gould[3] and Smiley,[4] the granulomata recurred and were removed two months and one month later, respectively, followed by cure.

CONCLUSIONS

These lesions are traumatic in origin, and in prophylaxis therefore, trauma is to be avoided. Tubes for intratracheal anesthesia should be made with a very smooth surface. They should be stiff enough to maintain an airway but sufficiently flexible to avoid trauma. The tube should be carefully selected for size for the individual patient and it should be gently introduced through a relaxed larynx.

Granuloma of the larynx following intratracheal anesthesia is most likely to arise from the vocal cord in the region of the vocal process of the arytenoid cartilage.

The removal of a pedunculated laryngeal tumor is easy and leaves a very small raw surface. It seems advisable, therefore, if the airway does not become dangerously inadequate, to allow these growths to become pedunculated before they are removed.

1403 DELACHAISE STREET.

REFERENCES

1. Clausen, R. J.: Unusual Sequela of Tracheal Intubation, Proc. Roy. Soc. Med., Section of Anesthetics, 1932, p. 1507.

2. Cohen, M.: Tumor of Vocal Cord Following Nasal Endo-Tracheal Anesthesia, Brit. M. J. 1:283 (Feb. 5) 1938.

3. Gould, R. B.: Laryngeal Granuloma Following Intratracheal Intubation, Brit. M. J. 2:499 (Sept. 14) 1935.

4. Smiley, W. A.: Polypoid Granuloma of the Larynx Following Endo-Tracheal Anesthesia, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 49:556, 1940.

XVIII

HAIRY OR BLACK TONGUE

MARGARET NOYES KLEINERT, M.D.

BOSTON, MASS.

A case of black tongue is presented because of the comparative rarity of the condition and because of the changing conception as to its etiology.

The interest in black or hairy tongue became greater when a question arose in regard to its parasitic or nonparasitic nature. Heidingsfeld[1] reviewed the literature up to about 1910. Before this 100 cases had been reported, mostly in French. As described by one French author, the discoloration under the microscope was light or dark brown, diffusely distributed over the hair. The hairs consisted of thickly crowded masses of long, thin epidermic cells with a tendency to branch. There were enlarged, cornified, pigmented, filiform papillae. A remarkable feature was the absence of fine granular masses of cocci, bacilli and leptothrix which are part of the normal coating of the tongue. No evidence of fungi was found by several of the French authors. One regarded it as hypertrophy of the filiform papillae with abnormal pigmentation and keratosis of the affected tissues. Others found micro-organisms abundantly present, a filiform bacillus, and round spores.

Heidingsfeld, in his article, reports the case of a man, 85 years old, who also had profuse salivation and pain referred to the ears. This patient developed a malignancy. It was considered a complication of the black tongue.

A second case reported by Heidingsfeld was in a boy of 19. It is described as a brownish discoloration of a triangular area extending from the circumvallate papillae anteriorly along the median dorsal surface of the tongue almost to its tip. There was "a soft, felt-like coating of matted hair on the processes." Pain was absent. The patient refused biopsy. During five years of observation there was no change. Some hairy processes were clipped off. No drugs were taken.

Some discoloration of the tongue in cases of syphilis has been observed by dermatologists, and it has also been noted in cases where

tobacco is used immoderately. These cases lack the clean-cut clinical characteristics of the two cases reported.

Microscopic examination is described in detail by Heidingsfeld. The histology of Case 1 is given as follows: "The filaments preserve for themselves a feather-like appearance. The filaments can often be traced to their origin from papilla-like projections of stratum corneum, the latter taking on a fringe of serrated cells."

Second in importance to the filaments is the formation of abnormal papilla-like bodies or epithelial nests, which Heidingsfeld calls "epithelial founts." Groups of degenerated cells are seen, which have the appearance of blood accumulations or blood cavities and are degenerated blood cells rather than pathogenic spores, which they resemble.

Leptothrix which shows a resemblance under the microscope has nothing in common, clinically, with hairy tongue.

Heidingsfeld considers there are two types of hairy tongue, the true and the false, described as follows:

True cases are characterized by well-defined, stable, brownish, thick, soft, fur-like patches covered by densely intertwined hair-like filament ⅛ to ½ inch long. The true cases owe their origin to an anomaly of development, perhaps of a congenital nature, being of stable character, remaining unchanged for indefinite periods and localized to definite areas. Such would not be the case if they were infectious or if they were inflammatory in nature.

The false cases are unstable and owe their origin to local irritation, infection, tobacco, antiseptics, or syphilis.

A case was reported by Weidman,[2] who reviewed the literature in 1928, of an obese white man, aged 63, who had had diabetes for 18 months. The lesion on the tongue was located in the classical position, on the posterior half of the tongue. It consisted of a poorly circumscribed, brownish patch, which might have been regarded as the effect of accumulated mucus and saliva stained by tobacco, which the patient used excessively. Only after the saliva was scraped away could the hairs be distinguished. When this was done, the lesion was found to be located in the circumvallate papillae and the hairs extended 10 mm. above the surface. They were dark brown, not black. The tongue elsewhere exhibited definite elevation of the conic papillae. All teeth had been extracted years before. The patient was unaware of the duration of this condition but thought it had been present for years. There was no discomfort. The hair was examined

bacteriologically, described and photographed. It was also examined histologically. No biopsy was done. Scraping dragged away the macerated keratinous substance which intervened between the hairs, which explains in part why the hairy appearance became marked on the patient's tongue after the removal of saliva. The culture was kept four months and finally showed hair-like lines and yielded short bacilli of slightly brownish color in the substrata.

Swinburne[3] did a biopsy in one case and an autopsy in another and describes the clinical features as follows: The condition is generally discovered accidentally by the patient or the physician. There is no reliable information from the patient as to its duration. Numerous cases may be observed in routine examination. The first impression is of a tuft of hairs on the dorsum of the tongue. The hairs are tough and withstand pulling. When cut off they leave, by contrast, a whitish area. Many cases are associated with inflammatory conditions, such as poor teeth, soft gums, new growths, tonsillitis. In some patients radium had been used to no effect.

He describes the bacteriology in one case as follows. The dorsum of the tongue was covered by a slimy mucoid deposit in which were lying long fine hairs. The mucus was acid. The culture grew yeasts. The mould black sporangium was found. Vibriones of a peculiar curved rod type were found in greyish-white colonies. The epithelium from which the hair-like processes were growing was hypertrophied. There was a dense growth of micro-organisms forming a mycelium in which were long chains with evidence of branching and clubbing at the ends. In one, there were long gram-positive bacilli of the B. subtilis or lactobacillus type. There were numerous irregular black pigment granules in the thickened epithelial layers.

It is difficult to adopt the suggestion that these organisms initiate the condition.

Swinburne does not agree in the pathological findings of Heidingsfeld.

The name "hairy tongue" seems a misnomer as the hairs seem to be a continuation of the filiform papillae.

About 1941 a change became apparent in the concept regarding the cause of the condition known as black tongue.

Layne and Watson[4] report a case of black tongue produced experimentally in dogs by a deficiency of nicotinic acid in their diet. They report that the greatest deficiency in vitamins seemed to be

nicotinic acid, although it is thought that in human pellagra there is a deficiency in all, or many, of the vitamins.

Little[5] reports a case of a fur-like tongue in a service man 24 years old. This man was a heavy smoker. No fungi were found. The case cleared in a short time under local treatment after he discontinued smoking.

Krehl[6] discusses the relative value of nicotinic acid in various foods and states that on dry weight basis carrots and tomatoes are the best vegetables as a source of this food property. All meats, yeast, peanut butter and whole wheat are excellent sources. Milk and eggs have a low nicotinic acid content. Whole cereals vary, the highest content of nicotinic acid being found in the bran of whole wheat.

Handler[7] discusses the effect of a cornmeal diet on dogs and the development of black tongue. He concludes that due to the inconsistent behavior of the dogs on the restricted nicotinic acid diet, it is difficult to evaluate the nature of the physiological defect.

Dann[8] working in the John and Mary R. Marble Foundation grant, states that further studies will be made, including one which examines the attacks on organisms invading the mucous membrane with chemotherapeutic agents.

Kennedy[9] reports three cases. He found monilia albicans and succeeded in obtaining the same organism as was found in two of the cases from some wood on which the patients had laid their cigarettes.

REPORT OF A CASE

Mr. A. W. C., aged 36, a lineman by occupation, was first seen in October 1944, complaining of an upper respiratory infection and hoarseness. At that time both antra were found dull by transillumination. The tonsils were absent. There was some pyorrhea but the teeth were good. No other abnormality was observed in the nose and throat examination.

On September 2, 1945, this patient came for examination complaining of a fullness in the throat and difficult breathing as if there was an obstruction at the back of the throat. The past history revealed that this patient had been working on a telephone installation in close proximity to a factory for weaving wool cloth. The patient averaged one and a half packages of cigarettes a day. His diet contained a high carbohydrate percentage. The patient had been operated on for gastric ulcer in 1943 and in 1944 for appendicitis and adhesions.

On examining the throat with a laryngeal mirror a mass of thick, brownish material was observed in both lateral ventricles, and there was an area in the center of the tongue covered by a whitish deposit. A brownish area extended up over the base of the tongue in a V shape. This area was covered by profuse, thick mucus, and saliva which revealed brownish hair-like papillae standing up when the area was scraped clean. But there seemed to be a quick return of the thick saliva in almost unending amount. The brownish hairs disappeared in a few minutes as the mucus collected again. Brownish stain was observed in the first mucus obtained of the scraping. After repeated scraping the mucus became clear but it remained copious. Time after time these brownish hairs were clipped off with curved scissors. More seemed to develop in the same area. A culture made at this time and examined at the Massachusetts Eye and Ear Infirmary showed many pleomorphic gram-positive rods. B. anthracoides, Bacteroides, alpha hemolytic Streptococcus were grown.

This patient was not ill; he stated he felt very well. After the preliminary discomfort in his throat he had no further local symptoms. There was no glandular involvement. The larynx was normal. The gums showed no pyorrhea.

The patient entered the New England Hospital on September 9, 1945. Physical examination was essentially negative with the exception of the tongue. He was a muscular man six feet in height, weighing 178 lbs. He received 1,100,000 units of penicillin sodium during his stay of five days. The urine examination was negative. The blood examination showed: hemoglobin 13.2 gm., erythrocytes 4.54, leucocytes 7,600, neuthrophiles 59%, lymphocytes 37%, large mononuclears 2%, eosiniphiles 2%.

A culture and a smear done at the New England Hospital on admission showed hemolytic staphylococcus albus, yeasts, B. subtilis, and a gram-positive bacillus, not identified further.

Sulfadiazine was administered by mouth. One gram was given every three hours during the last three days of hospitalization. Vitamins were given continuously three times a day. Local examination and treatments were done each day. The tongue appeared to be lighter in color, but never cleared entirely. He was discharged from the hospital September 14, 1945. Penicillin troches, 1,000 units, penicillin tablets, 25,000 units, vitamins, and penicillin nose drops were given to be used as home treatment.

Throughout hospitalization the temperature level was 98.6° F., pulse 80, respiration 20.

The culture made before discharge from the New England Hospital showed B. coli communis, yeasts, hemolytic staphylococcus aureus.

During the next two weeks the patient was observed and treated several times by local applications to the tongue and throat. As the tongue had soon returned to its original darkness and roughness, it was decided to give a second series of penicillin treatments. The smear and culture during this interim showed an increase in the usual throat flora of streptococci and staphylococci.

The patient re-entered the New England Hospital October 2, 1945. At this time the same treatment as at the previous admission was given. A total of 1,190,000 units of penicillin was given in seven days. A total of 42 gms. of sulfadiazine was given. The blood level of sulfadiazine was 8.7%. The other laboratory tests showed a total protein of 6.7. The Hinton test was negative. The blood count, temperature and urine were normal. The report on the smear and culture was: "No fusiform bacilli or spirochetes present. Mycelium and yeast cells present in clumps, also thread-like material. Large squamous epithelium cells, debris and mucus. A moderate number of bacilli and other miscellaneous organisms."

The examinations of the tongue before discharge showed no permanent improvement in the condition after seven days of penicillin and sulfadiazine treatment. During this time other agents, such as perborate gargles, penicillin troches, 1000 units each, and sulfanilamide throat tablets, were used. Daily cleaning of the dark area by curettage and applications of various antiseptics were done to no avail. The patient was discharged from the hospital after seven days of intramuscular penicillin treatment with not much improvement in his condition.

As the recent literature associates black tongue with pellagra and nutritional deficiencies,[5, 7-10] the patient, on discharge, was given a high vitamin diet, and cevitamic acid, 100 mg. tablets, vitamin D, 100 mg. tablets, and vitamin B complex tablets, one of each a day.

DISCUSSION

A patient was hospitalized twice for treatment of a condition known as hairy black tongue. At the first bacteriological examination B. anthracoides was found. Penicillin was given intramuscularly every three hours for about ten days. The tongue was given daily treatments which consisted of scraping it as clean as possible with a large spoon curette. By holding the tongue out and using a laryngeal

mirror it was possible to visualize the large base of the darkened triangle. This seemed to be heavily coated with an adherent whitish material about the consistency and appearance of cream cheese. It never seemed possible to get it all off.

During the few treatments when the patient was first seen before he entered the hospital, at the lateral aspects of the tongue the pyriform sinuses were filled with brownish moss-like masses.

The penicillin injections were supplemented by sulfadiazine. At the end of the fifth day the tongue appeared cleaner, although not normal as there was a brownish tinge remaining which could be roughed up by scraping.

As it was not considered desirable to keep the patient hospitalized longer at this time, he was discharged but he was to continue local penicillin treatment. The culture made at the time of discharge did not show any of the anthraxlike bacilli which were present on the first examination. There still were streptococci and staphylococci.

A week after the penicillin injections the tongue appeared to be getting worse again. There was about the original amount of dark brown color and debris which could be scraped off. There were profuse stringy mucus which had a brownish tinge and hair-like projections of the papillae. These could be cut off as before. There were no more collections of moss-like masses in the pyriform sinuses. This was no doubt due to the frequent throat washes and better mouth hygiene.

From a review of the literature it would seem that hairy black tongue in itself is of no significance. When complicated by a superimposed infection it calls attention to itself. In the case reported here a bacillus belonging to the anthrax family was isolated on the first bacteriological examination and immediate penicillin treatment was started. Penicillin treatment does not eliminate the condition known as hairy black tongue but it returns the throat to a more normal condition.

No attempt at a complete bibliography of the early literature is made as that was done in the comprehensive review by Heidingsfeld[1] in 1910, and by Weidman[2] in 1928.

CONCLUSIONS

Treatment with penicillin injections and sulfadiazine in the case reported of hairy black tongue proved ineffectual. Four months after

instituting a higher vitamin diet and vitamins, especially the recommended nicotinic acid, there has not been appreciable improvement.

23 Bay State Road.

REFERENCES

1. Heidingsfeld, M. L.: Hairy or Black Tongue, Report of 100 Cases, J. A. M. A. 55:2117 (Dec. 17) 1910.

2. Weidman, F. D.: The Affinity Between Black Tongue and Trichomycosis, Arch. Dermat. & Syph. Vol. 18 (Nov.) 1928.

3. Swinburne, George: Black Hairy Tongue, J. Laryng. & Otol. 54:386-404 (July) 1939.

4. Layne, J. A., and Watson, C. J.: Pellagra In Dogs, Ann. Int. Med. 19: 200-205 (Aug.) 1943.

5. Little, R. D.: A Case of Black Tongue, U. S. Nav. M. Bull. 43:360-361 (Aug.) 1944.

6. Krehl, W. A., Teply, L. J., and Elvehjem, C. A.: Effect of Corn Grits on Nicotinic Acid Requirements of the Dog, Proc. Soc. Exper. Biol. and Med. 58:334 (Apr.) 1945.

7. Handler, P.: Use of Highly Purified Rations in the Study of Nicotinic Acid Deficiency, Proc. Soc. Exper. Biol. and Med. 52:263 (Apr.) 1943.

8. Dann, W. J., and Handler, P.: Nicotinic Acid and Coenzyme Content of Tissues of Normal and Black Tongue Dogs, J. Nutrition 22:409, 1941.

9. Kennedy, C. B., and Knowles, J. K.: Black Hairy Tongue (3 Cases), Arch. Dermat. & Syph. 42:566-569 (Oct.) 1940.

XIX

FIBROLIPOMA OF THE FACE

REPORT OF A CASE

D. F. WEAVER, M.D.

DETROIT, MICH.

A review of the literature indicates that fibrolipomata involving the face are rare.

Mena[1] distinguishes between deep and superficial, and congenital and noncongenital tumors.

He mentions a case in the following translation: "Le Lentu, quoted by Deulafe, mentions a healthy female child who presented diffuse tumor of the left cheek and half of the lips. The adipose tissue which constituted it was very adherent to the deeper parts of the skin, and the face gave the appearance of a hemihypertrophy."

It is felt that the following case is of interest, first because of the relatively infrequent occurrence of this type of tumor and secondly because of some of the technical difficulties involved in its removal.

A 29-year-old, married, white woman was admitted to the Henry Ford Hospital on May 23, 1945. She stated that at 13 years of age she had first noticed a swelling in the region of the right cheek bone and eye. This had enlarged very slowly until five years before admission, when enlargement had become more rapid and she had noticed decreasing vision in the right eye. There had been moderate right frontal headache. It had been necessary to shave the hair from a large area over the right parotid region frequently.

On examination, a soft movable tumor was present on the right side of the face. (Fig. 1.) This involved both eyelids, and the malar, temporal, and parotid regions. In several areas, including each lid, there could be felt firm movable tumors within the soft tumor. These varied from 1 to 3 cm. in diameter. A large bone defect could be palpated in the region of the right orbital margin. Hair grew from an area 4.5 x 5.5 cm. over the right parotid region. Roentgenograms showed evidence of a bone defect along the lateral and superior margins of the right orbit and also a defect in the right frontal bone running

Fig. 1.—Preoperative appearance showing tumor and hair bearing skin.

upward and backward. There was also evidence of the soft tissue tumor. (Fig. 2)

Examination of the ear, nose and throat was negative. Vision was 20/32 in the right eye and 20/20 in the left.

The general physical examination revealed no significant abnormalities. The systolic blood pressure was 110 mm., the diastolic 75 mm. The hemoglobin was 13.9 gm., the red blood cells 4,390,000 per cu. mm., the leukocytes 4,400 per cu. mm. with 69% polymorphonuclears, 29% small lymphocytes, and 1% eosinophiles and 1% mononuclears. The Kline exclusion test was negative. The examination of the urine was negative.

On May 25, 1945, under nitrous oxide-ether anesthesia, administered by the intrathacheal route, an incision was made extending from back of the hair line in the temporal region to about 3 cm. above the angle of the jaw. This ran close to the ear in the parotid region. The under surface of the tumor separated readily from the parotid fascia but elsewhere it was firmly attached and extremely vascular. It appeared to infiltrate the skin and subcutaneous tissue, consequently it was removed by sharp dissection throughout, an attempt being made to leave a sufficient thickness of skin to assure its proper healing. A

Fig. 2.—Roentgenogram showing bone defect along lateral and superior margins of the right orbit and in the right frontal bone. The margin of the soft tissue tumor can be made out.

large part of the hair bearing skin was excised. A soft rubber drain was inserted and a pressure dressing applied.

The extreme vascularity of this tumor made it impossible to proceed without troublesome active hemorrhage. Consequently, in addition to 1000 cc. of 5% glucose and 500 cc. of plasma, which were given during the operation, it was necessary to give one liter of whole blood within a few hours following the operation. The patient did not take fluids well by mouth for several days, so fluids were given intravenously.

On the third post-operative day the sutures were removed but since there was moderate drainage of serum, the drainage tube was left in place until the fifth postoperative day.

The postoperative course was otherwise uneventful and the patient was dismissed from the hospital on the eleventh postoperative day, at which time the hemoglobin was 11.7 gm. and the red blood cells 3,660,000 per mm. She was given 15 grains of ferrous sulphate daily.

The pathological diagnosis made on microscopic examination of fixed sections of the tumor was fibrolipoma.

The patient returned to the hospital on July 17, 1945, at which time examination of the blood showed no evidence of anemia. The skin of the upper lid was excessive and a firm nodule about 1 cm. in diameter could be palpated in the orbit beneath the lower lid. This had apparently been overlooked at the previous operation.

On July 20, 1945, after local infiltration of 2% novocaine, an elliptical strip of skin about 15 mm. in width was excised transversely from the upper lid. The nodule was removed from below through this incision and the wound closed with fine interrupted silk sutures which were removed on the third postoperative day. The patient left the hospital on the fifth postoperative day.

A refractive error was corrected.

Eight weeks later the result appeared to be completely satisfactory. The function of both lids was unimpaired.

HENRY FORD HOSPITAL.

REFERENCE

1. Mena, R.: Fibro-Lipoma del carrillo, Arch. Cubanos Cancerol 3:102-104 (Apr.-June) 1944.

XX

TWO UNUSUAL ESOPHAGEAL CASES

ARTHUR C. JONES, M.D.

BOISE, IDAHO

All peroral endoscopy is fraught with some danger and the examining surgeon should have had preliminary, careful and complete training under the guidance of an expert before attempting any type of endoscopy. Recently I have seen three cases emphasizing this—two were in children with generalized emphysema of the neck and perforation of the trachea and a rapidly ensuing death followed bungling endoscopy.

CASE 1.—The case I am reporting is of Mrs. S. A., age 46, edentulous, who gave a history of pain in the esophagus while eating pork chops. She saw her family doctor who used the dangerous, antediluvian probang.

One week later she came to my office with a temperature of 101.8° F., marked substernal pain, somewhat dehydrated and having the appearance of being a very sick woman. She had lost ten or twelve pounds during the preceding week and had had almost continuous pain, particularly beneath the sternum, which radiated into both ears after attempting to swallow.

She was sent to the hospital where penicillin, 25,000 units every three hours, was started immediately. The radiologist was called and his report is as follows: "Lateral film of the esophagus shows marked swelling of the soft tissue behind the larynx and trachea. This soft swelling measures four cm. in its anterior posterior diameter. There is a tiny fragment about two by five mm. which has the appearance of a foreign body lying in this soft tissue density anterior to the sixth cervical segment. Fluoroscopic examination with barium shows considerable spasm in this region with a small fleck of barium remaining in the region of the foreign body."

I might say in passing that Vinson,[1] condemns the use of barium. He states: "If a foreign body is present barium may cover it so completely, especially if it is a bone, that the difficulties of esophagaic

---

Presented before the Western Section of the American Laryngological, Rhinological and Otological Society, Los Angeles, Calif., Jan. 26-27, 1946.

removal are increased to a marked degree and that the administration of barium paste may complete the obstruction if there is already some obstruction." He suggests the use of iodized oil. Fortunately up to the present time I have had no untoward experiences or accidents after the use of barium.

Because of the fact that the patient was dehydrated, and because of the marked swelling of the esophagus, I thought it advisable to continue the use of the penicillin during the night along with intravenous administration of 1,000 cc. of 20 per cent glucose in sodium chloride solution.

The next morning the patient appeared in better condition. Under a general anesthetic the esophageal examination was made. Just below the cricopharyngeus I saw a fairly large piece of bone directly across the lumen of the esophagus. All of the bone could not be seen as it had apparently penetrated the esophagus. This piece of bone was carefully removed. There was considerable pus around the bone and a marked odor came up through the esophagoscope. Three other smaller pieces of bone were removed.

This was all done under complete general anesthesia. I am quite firmly convinced that a complete general anesthetic for the removal of foreign bodies of the esophagus is much safer for most operators than an attempt to remove the foreign body under local anesthetic with the accompanying spasm of the esophagus.

Gastrostomy is frequently advocated in patients with marked dehydration. This operation in the dehydrated individual has a mortality rate of 10 to 50 per cent and I much prefer using 1,000 to 2,000 cc. of a fluid containing 20 per cent glucose.

The evening following removal of the foreign bodies the patient's temperature had dropped to 100° and most of her substernal pain was gone. The penicillin was continued. She was allowed to drink a half glass of water containing ¼ teaspoonful of bismuth powder at rather frequent intervals.

She continued to have some mediastinal discomfort and two days later the radiologist reported: "Further lateral film of the esophagus shows no definite evidence of an opaque foreign body. Fluoroscopic examination, after swallowing barium, shows a large ulceration about two cm. in diameter which is connected with a very narrow neck to the lumen of the esophagus. This ulceration appears to be in the soft tissue posterior to the esophagus and represents a perforation, and barium can be seen leaving the esophagus."

Two days later the patient's temperature was normal, her substernal pain had disappeared and she was anxious to leave the hospital. However she was kept on penicillin for ten days. Her food intake was limited to liquids consisting of soup and gruel.

Re-examination of the esophagus nine days later showed: " . . . the previously described perforation to have closed. Barium no longer leaves the esophagus through the perforation and the cavity which was previously present is no longer seen. There is no spasm or ulceration in the esophagus."

The patient was allowed to go home, cautioned to refrain from eating heavy solid foods for the next week and in the following month had gained back her lost weight.

CASE 2.—Mrs. A. H., age 60, edentulous. I was called in consultation to see this lady. She gave a history of choking while eating a beef steak ten days previously. She had had considerable substernal pain and had been running a temperature of 99° to 100° F. She was able to swallow fluids but there was considerable pain attached to the act of swallowing.

The radiologist's report is as follows: "Studies of the chest and esophagus show no evidence of an opaque, foreign body in the esophagus. There is a crater one cm. deep on the right lateral wall of the esophagus in the region of the third dorsal segment. There is an intense spasm with marked edema of the esophagus at this point." His diagnosis was ulcer of the esophagus following the ingestion of a foreign body.

Occasionally it is very difficult for the radiologist to be absolutely certain that there is or is not a foreign body present in the esophagus. Because of her marked discomfort and dysphasia I was tempted to examine her with the esophagoscope, but because of the large ulceration of the esophagus I was fearful that any untoward manipulation might cause a rupture of the esophagus. Consequently I decided to watch the patient for a few days.

She was getting penicillin, 25,000 units every three hours. Her symptoms and complaints subsided in about three days and 13 days later the radiologist reported: "Further studies of the esophagus show the previously described condition to have almost entirely disappeared. There remains a small amount of spasm with shallow mucosal excavation about two mm. deep."

She was allowed to leave the hospital and has completely recovered.

Comment. The report of one case of definite perforation of the esophagus cured without the patient developing a mediastinitis is not sufficient to draw any positive conclusions but if we can prevent the necessity for the operation for mediastinal abscesses, which is itself not without danger, and the meticulous aftercare that is necessary to save the patient's life, I think it is good judgment to start the potential mediastinal case on penicillin immediately.

In the past the thoracic surgeon has thoroughly emphasized the necessity of very early mediastinal drainage, wherever there was evidence of esophageal perforation. The earlier the surgery the lower was the mortality. But now with the advent of chemotherapy this formidable procedure can at least be delayed.

In a personal conversation with Dr. A. C. Furstenburg he states that he has had a similar experience in a case of esophageal perforation.

The fact that we have this safeguard, chemotherapy, to fall back upon does not decrease the necessity of skilled, aseptic surgery in all types of operations.

I am indebted to Dr. Alfred Popma, radiologist for St. Luke's and St. Alphonsus Hospitals of Boise, Idaho, for his help and advice in Case 1.

518 EASTMAN BUILDING.

REFERENCE

1. Vinson, P. P.: Diagnosis and Treatment of Diseases of the Esophagus, Springfield, Ill., Charles C. Thomas Co., 1940.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, November 5, 1945

THE PRESIDENT, DR. JOHN F. DELPH, IN THE CHAIR

The Significance of Positional Nystagmus In Otoneurological Diagnosis.

JOHN R. LINDSAY, M.D.

(Abstract)

Clinical observations on postural or positional vertigo and nystagmus were made in a series of approximately 100 cases of so-called "pseudo-Ménière's syndrome." In this sydrome the absence of auditory disturbances and other neurological findings has made the localization of the source of the vertigo indefinite. Both the examination for spontaneous nystagmus in the customary upright position and the routine tests for vestibular function have usually failed to indicate definitely the location of the disturbance. On the basis of reports by former observers, particularly those of Nylen on brain tumors and Seiferth on other otoneurological conditions, it has been possible to differentiate two simple types of postural or positional nystagmus:

Type I.—the nystagmus changes its direction with changes in position or while the head remains in the same position. Type II—the nystagmus maintains a constant direction but it may only appear in certain positions or is influenced in intensity by changes in position.

Type I positional nystagmus has been found only in lesions involving the central vestibular pathways, whereas Type II positional nystagmus has been found in both peripheral and centrally located lesions.

In the so-called pseudo-Ménière's syndrome, vertigo which is postural in type is a characteristic finding. The postural nature is

sometimes obscured during the severe stage of the attack but becomes apparent as the vertigo begins to subside, or it may be characteristic from the beginning of the attack. The observations in this series indicate that the positional nystagmus in pseudo-Ménière's syndrome is of Type I in the great majority of cases. Type II has also been found in a smaller number. On the basis of information gained from proven lesions both of the peripheral and central vestibular apparatus, that Type I positional nystagmus is an indication of a lesion affecting the central vestibular apparatus, this type of nystagmus in pseudo-Ménière's syndrome is probably good evidence of a central origin. Type II positional nystagmus, which is sometimes also seen in pseudo-Ménière's syndrome might arise from either a peripheral or a central location in such cases.

Approximately one-half of the cases of pseudo-Ménière's syndrome consisted of patients with an unusually low blood pressure and other symptoms of vasomotor instability, sometimes called the hypotensive group. In the other half the vertigo could be attributed to such causes as toxic disturbance, and hypertension, and in many cases the etiology was indefinite.

DISCUSSION

DR. SHERMAN L. SHAPIRO: As Dr. Lindsay's paper is a clinical one perhaps the most useful comment I can make is to summarize my own experience. This comprises probably more than 1000 patients in whom I have observed this finding. Most of them have had head injuries, but there have also been a good many cases of pseudo-Ménière's syndrome. I have used Frenzel glasses for more than twelve years and consider them indispensable, because without their use I am certain a number of cases of mild degree of postural nystagmus would have been missed. In my experience this has been the most frequent objective finding where vestibular abnormalities were present. The most common form has been the horizontal and the most frequent direction, to the left.

Excluding medicolegal cases, my experience has been that when patients complained at the time of examination, a study with Frenzel glasses invariably showed some degree of postural nystagmus as well. I believe both phenomena are of vestibular origin and, in the great majority of cases due to central nervous system involvement. I do not see how a peripheral disturbance can be diagnosed in the absence of marked hearing loss or of otitis media. There are in my experience two types of postural nystagmic reaction; in one the nystagmus does not occur immediately, but after a lapse of a few seconds, and is pro-

nounced. In the second variety it comes on at once and is weaker both as to amplitude and duration. It would seem that the mechanism of occurrence in these two varieties is somewhat different, but to say that the first type, or true postural nystagmus, is due to otolithic involvement is to take a good deal for granted, particularly as no one has produced nystagmus by otolithic stimulation. I have not been able to classify my cases as to types, possibly because my material included relatively few tumors. Most of the people I have seen showed Type II nystagmus, yet the great majority have unmistakably had lesions of central origin. Toxic and irritative factors, mentioned by Dr. Lindsay, are of course important, but more important in my experience are circulatory changes, that is, arteriosclerosis, hypertension and, in head injuries, vasomotor changes. In nearly all of the last named I have found a postural nystagmus at the outset. However, if the patient is followed, the nystagmus is seen to disappear later in most instances as the vasomotor centers take control. There is no doubt in my mind that the underlying disturbance is a transient anemia or hyperemia which momentarily affects the vestibular center.

I think we have been privileged to hear a timely and very able discussion of this important subject which will be of practical benefit to every otologist.

DR. E. J. BLONDER: Dr. Lindsay has been very courageous in taking up this subject. A few years ago I went over the literature quite thoroughly and it contains many controversial reports. Otologists from Bárány on often speak of positional nystagmus as being of semicircular canal origin. Physiologists and those doing actual experimental work as a general group consider positional labyrinthine reflexes due to otolithic stimulation. However, otolithic reflexes have never been demonstrated experimentally to produce nystagmus.

The reason there is much clinical discrepancy in the literature is that there is relatively little experimental work on lesions of portions of the labyrinth and the correlation of these lesions to physical findings. There are great technical difficulties in producing these isolated lesions in a live animal. Versteegh reported section of the utricular nerve in only one rabbit. Magnus and DeKleyn did some fine original experiments on positional labyrinthine reflexes.

As to Dr. Lindsay's method of noting positional nystagmus, the physiologists would not be in accord with his method of recording nystagmus immediately after the change in position, but would prefer to note the reading after a short interval of time, to make sure

the reflex observed was due to position, not to the motion caused by changing position. Examining a large number of patients with verified intracanial lesions, I did not record anywhere near 90 per cent with positional nystagmus; on the contrary, only a very few responded with positional nystagmus. I had the opportunity to review labyrinthine tests in histories in a number of institutions, and attempted to correlate the labyrinthine tests with operative intracranial findings. One I have in mind was a tumor of the pons; five or six years previously, an otologist had examined that patient and found no nystagmus upon ordinary caloric stimulation, but was able to bring out nystagmus with irrigation of the ear with alternating hot and then cold water. There is a question whether that type of observation is of much clinical value in diagnosis. To me positional nystagmus alone has not much significance in localization unless an otoneurological examination, or a neurologist expert in brain pathology, can bring out additional findings which may be correlated in arriving at a diagnosis. Otherwise I would just note that the patient has nystagmus on certain positions of the body. If a patient were toxic or had vasomotor disturbances, how could we be sure whether the effect was on a central or peripheral location?

DR. ALFRED LEWY: I would like to ask Dr. Lindsay whether he has correlated the caloric reaction with the postural nystagmus findings? Is there any constant relationship? In the case of hypotension was the blood pressure taken before and after the test? I have seen two cases of organic heart disease in which I believe hypotension caused the vertigo. We could not prove it, of couse, except therapeutically; therapeutic measures to improve the heart action improved the vertigo.

I should like to have this dissertation presented to some industrial surgeons. Some are inclined to believe all disturbances are due to psychoneurosis or malingering or a desire for compensation. That question is sometimes very difficult to answer, and I wonder if Dr. Lindsay ever found objective evidence, such as postural nystagmus, in cases of pure psychoneurosis. I disagree with Dr. Blonder in the idea that unless the neurologist can spot the otoneurological symptoms there is nothing to it. I know of a number of cases in which otoneurological symptoms were the only objective ones present in organic changes in the brain.

My idea that industrial surgeons should avail themselves more frequently of the services of the otologist before sending back to work a man with a head injury is based on a number of experiences. One of the large railroad companies elected to take the word of their

chief surgeon in such a case, and the man was put back to work, perhaps with the idea that he was suffering from merely a psychoneurosis. He quite promptly fell off a freight car and sustained another head injury with unconsciousness, and the railroad paid a very substantial sum in settlement of the case. They had had the advice of an otologist who did not agree with the chief surgeon, but they elected to take the latter's opinion.

Dr. Howard C. Ballenger: I would like to stress one point in the etiology of pseudo-Ménière's disease. In my experience in ordinary office practice in which acute upper respiratory tract infections and their complications form a large part, acute infections or congestion of the middle ear and the eustachian tubes seem to be the most frequent cause. In a large clinic other etiologic factors, such as circulatory disturbances, would probably be found most frequently.

Dr. John Lindsay (closing): I wish to repeat the primary object of this study, which was to try to determine, if possible, whether postural or positional nystagmus contributes reliable information which will help to differentiate a peripheral from a central origin of the vertigo. During these observations a Type I positional nystagmus has not been observed in any known peripheral lesion, that is, one in which the vertigo was associated with tinnitus or deafness. All positional nystagmus of known peripheral origin was Type II. In the pseudo-Ménière's group a postural vertigo and positional nystagmus proved to be an almost constant characteristic, although sometimes obscured in the more severe stages. Type I occurred in the majority but Type II also occurred in a few.

Evidence obtained from known brain lesions indicates that Type I positional nystagmus occurs mainly in pontine and cerebellar lesions. Some tumors above the tentorium will also show postural nystagmus. Nylen has noted a vertical nystagmus in the head-hanging position in such cases.

Many of the patients examined had a spontaneous nystagmus in the upright position during some stage of the attack. The postural character may become evident in subsiding stages or may be characteristic throughout.

Regarding the question as to whether a certain toxic disturbance without other neurologic findings can be said to be central rather than peripheral in origin, it can be said that on the basis of the factual information accumulated so far it looks as though Type I positional

nystagmus is a reliable indication of a central origin and the majority of the pseudo-Ménière's group have had this type.

Frenzel glasses are designed to eliminate the possibility of the patient fixing his gaze on objects in the environment and thereby suppressing nystagmus. They also give a better view of the eyeball.

No definite relation has been observed between the caloric responses and a postural nystagmus. In the cases of pseudo-Ménière's syndrome the caloric reaction was routinely within normal limits. In posterior fossa tumors there were all sorts of variations in the response. For instance, the caloric response may be absent on one side before operation and present again after operation, whereas the positional nystagmus may be present before and absent after operation in the same case. There is no definite correlation that we have been able to observe.

The relationship of blood pressure to vertigo is an interesting question. It is known that there is a vasomotor response from stimulation of the labyrinth and the question might be raised as to whether low blood pressure is not due to vertigo. But it seems, so far as we could tell, that the low blood pressure was fairly constant throughout the period that the vertigo could be elicited.

I do not think that routine neurologic and otologic examinations have taken sufficient cognizance of postural nystagmus. It is one of the most useful tests for a vestibular disturbance. I have never observed a positional nystagmus due to psychoneurosis. The presence of such a nystagmus with its accompanying vertigo is probably a reliable indication of a true vestibular disturbance.

We have seen some cases, not many, of vertigo due to nonsuppurative middle ear disease, but these are classified under "Ménière's syndrome" because they routinely have some interference with auditory function. In our experience this has not been a large group.

The Relation of Eye Movements to the Vestibular System in Man.

HENRY PERLMAN, M.D.

(Abstract)

The otologist is largely dependent on the studies of spontaneous and induced eye movements in evaluating vestibular function. Because of this a number of points are worth emphasizing.

1. The teaching of vestibular eye movements should include a consideration of all mechanisms involved in eye movements and the common lesions of all these systems that distort normal movement.

2. Because of the higher cortical organization of the visual system in man and the introduction of such factors as consciousness, volition, attentions, discrimination, etc., the clinician should study this problem in man. By careful observations and recordings, correlated with other neurological findings and postmortem or operative findings, conclusions arrived at can be valuable. In the words of the late Gordon Holmes, the method used should be the same as that of an experimental physiologist: to study the simpler components of a complicated function, isolating each of them and then attempting to integrate them into a complete picture.

3. The moving picture record of pathological eye movements is valuable when properly interpreted and can bring to the student in a short time the results of an extensive clinical experience. The moving picture can also demonstrate for comparison all normal movements.

4. The method used in this study for recording eye movements in man has certain advantages to the otologist wishing to investigate some of the problems in this field. Among them are the facts that it does not encumber the eye, it can record movement of the closed eye, and it can be used with the patient on a stretcher.

The organization of the subject of the basic eye movements in man is attempted in a moving picture so that one may use it as a framework of reference for interpreting abnormal movement. This organization is assisted by using a number of general theoretical concepts. Nevertheless, lesions of the brain stem and the midbrain particularly will continue to tax all our understanding. The result of a central lesion is not simple loss of function but the response of the remaining neural apparatus to the abnormal situation produced by the lesion, including disordered activity of the undamaged structures. Instead of "nystagmus in direction of gaze" we use the expression "inability to maintain lateral deviation." We assume that the slow movement toward center is due to tonic muscle pull tending to keep the eye at center and to return the eye to center when it is moved away from center. The quick return movement to the deviated position is considered the result of cortical stimuli that are adequate to move the eye but are inadequate to maintain the eye in deviation. Again, as the cortex releases the eye and allows it to start sliding back slowly toward center, the cortical stimuli again become

adequate and produce another quick movement. When the cortex releases the eye, as in anesthesia, coma or sleep, the eye assumes a more or less central position. When the eye is moved away from center mechanically it slides back to center when released, indicating an underlying tendency for the eye to return back to center— slowly— when released by the cortex.

One may further consider that the central position of the eye is due to symmetrical tonus of the eye muscles and that this symmetry may be disturbed by brain stem and cortical lesions, resulting in an eccentric zero position for reference of movement.

Further, in the presence of spontaneous vestibular nystagmus, more marked eye movements are observed when the subject looks toward the quick component than toward the slow component. This may be explained by considering a super position of the slow vestibular pull on the slow tonic pull toward center. The addition of these two similarly acting stimuli results in a stronger movement for the cortex to overcome in maintaining deviation; hence more periods of inadequacy of the cortex, interspersed with periods of recovery. In the directing of the slow component a cancelling out of the two factors producing slow movement toward center leaves the cortex better able to maintain deviation. When the frontal cortex is not adequate, the reflex retinal system producing fixation becomes accentuated. This modifies eye movement.

The method for recording eye movements is explained and the results of some experiments described and illustrated by lantern slides.

The organization plan of the movie is described, starting with the eye in central position, free of cortical control.

DISCUSSION

DR. SHERMAN L. SHAPIRO: There was an apparatus, used, I believe, by Benjamin, for measuring movements with glasses, consisting of occluded glasses with markings on them, something like the Bartels glasses. If the contra-rolling movements take place with fixation out of the reckoning, how can Dr. Perlman say it is due to occipital reflex?

DR. HENRY PERLMAN (closing): The first work on this problem was done by physiologists and psychologists who demonstrated that it was possible to use such a method for recording eye movements. It seemed an opportunity for otologists to study some of the clinical problems in this field using the new method.

The apparatus we used was an A.C. condenser coupled amplifier with a very wide frequency response range and a high gain; 60 microvolts produced about a quarter-inch displacement of the pen. When we record with an ink writer we are of course limited by its physical properties. But the resonance frequency of the ink writer was about 100 per second, quite adequate for recording these relatively slow eye movements. There is one fault with this kind of amplifier; the displacement of the needle is dependent upon the decay of the condensors. The time constant represents one-half the period for total decay and is about .8 second. As you noted in the records, the eye in the maintained deviated position does not keep the pen of the ink writer constantly deviated from center. The pen gradually recedes toward center. That represents the decay of the amplifier. However, practically all the phenomena we wish to record are faster than .8 second.

All these records were made with the assistance of Dr. Case, who is in charge of the laboratory of electro-encephalography. From our records it appears that regular counter-rolling movements of the eye do not occur with the eye closed. The eyes behave differently when opened even behind strong lenses. As long as there is retinal stimulation something happens that makes the open eye behave differently from the closed eye. We feel sure we are dealing with obliteration of the retinal system when the eye is closed.

Abstracts of Current Articles

NOSE

Management of Fractures Into the Nasal Sinuses

Shea, John Jr.: *Laryngoscope* 56:22-25 (Jan.) 1946.

Proper management of compound fractures of the maxilla includes roentgenologic studies although Shea feels that an experienced surgeon can anticipate the x-ray findings.

He advocates early reduction of fractures in cases not complicated by intracranial injury and this can usually be accomplished rather simply through an exposure of the anterior antral wall in the canine fossa. The malar bone may be pushed forward by proper manipulation of a heavy elevator and a depressed zygomatic arch may be sprung laterally by the same means. These maneuvers are often necessary to provide ample clearance for the coronoid process of the mandible.

Inspection of the interior of the antrum may be necessary and the cavity may require a tight gauze pack in cases of fractures in the pterygomaxillary wall or in the floor of the orbit.

Shea makes use of an antral balloon to keep the sinus roof in place. This is inserted through a naso-antral opening in the inferior meatus. The balloon is filled with air or water and kept in place from five to seven days. Uniformly good results are obtained and the procedure often obviates the necessity of wiring and use of complicated external appliances.

VAN ALYEA.

BRONCHUS

Bronchoscopy: Its Application in the Diagnosis and Treatment of Bronchopulmonary Disease.

Jeria, M. M.: *Revis de Otorrlaring.* 5:122 (Sept.) 1945.

The author relates his experience in 483 bronchoscopies on 236 patients with different forms of bronchopulmonary disease.

In suppurative pulmonary disease bronchoscopic study and repeated aspirations combined with medical treatment and surgery

when necessary resulted in recoveries in 15 per cent of cases and marked improvement in 33 per cent of cases.

A patient with bronchiectasis can be greatly relieved by aspiration.

The aspirated secretions should be studied in each case. Mycotic infections are readily diagnosed by examination of smears and might otherwise be missed.

Cystic disease is amenable to aspiration and treatment carried out through the bronchoscope.

Bronchoscopy is essential for location of fistulae and is an important aid to the surgeon during operative procedures.

HIGBEE.

Diagnosis and Treatment of Foreign Bodies in the Upper Respiratory and Digestive Tracts, also Complications Caused by Them.

Barrets, P. de M.: Rev. Brazil de Otorinolaring. 12:221-83 (July) 1944.

This author discusses the symptoms resulting from foreign bodies, methods of treatment and the value of x-ray in diagnosis.

Foreign bodies lodged in the respiratory and digestive tracts will produce symptoms varying with the levels of obstruction or inflammation.

Cough is most frequently the first symptom and it is of great importance in the history. It establishes the probable time of aspiration. There is frequently a lapse of time varying from hours to weeks or months after this initial symptom during which the patient is free of symptoms. As an example of this, a case is cited in which a patient developed a cough which was typical of whooping cough, but proved to be due to a nail aspirated five years previously and lodged in the right main bronchus.

Morphine and scopolamine are given in preparation of the adult patient for removal of foreign bodies. Frequently the author has found that as a result of this, foreign bodies lodged in the hypopharynx or esophagus have passed on to the stomach before the scope was inserted.

If possible, it is better to delay immediate removal if previous unsuccessful attempts have been made. This is especially true if fever is present, as it gives time for sulfonamide therapy.

If edema is present in the pharynx or the esophagus, it may be diminished by applications of adrenalin and local anesthesia. This condition calls for great caution in advancing the instrument.

If during removal of a foreign body the mucosa is perforated, it is necessary to enlarge the area sufficiently to provide free drainage and prevent the formation of an abscess. Some suitable antiseptic should be applied freely at the time. An examination should be made two days later and the condition noted. If the perforation has healed the patient may be discharged, but if it has not, then a feeding tube is inserted and full doses of sulfonamides are begun. Satisfactory internal drainage cannot always be accomplished and a cervical mediastinotomy may be necessary.

In a series of 600 cases no emergency tracheotomies were done on the author's service. Tracheotomy had been done on three patients before admission. In two of these there were several foreign bodies present which obstructed both main bronchi.

HIGBEE.

ESOPHAGUS

Esophageal Lesions Associated with Acrosclerosis and Scleroderma

Olsen, Arthur M., O'Leary, Paul A., and Kirklin, B. R.: Arch. Int. Med. 76:189 (Oct.) 1945.

The authors report on the incidence of esophageal symptoms and esophageal lesions occurring in patients on whom a diagnosis of scleroderma or acrosclerosis had been made, at the Mayo Clinic between the years 1930 and 1943. In 36 of approximately 350 such cases, dysphagia occurred, or lesions of the esophagus were demonstrated. They are reporting primarily on 18 cases in which conclusive roentgenographic or endoscopic observations were made.

In 15 cases the conclusive roentgenographic features demonstrated were esophageal dilatation with atony of the musculature, apparent spasm at the cardia with obstruction, and esophageal hiatal hernia. In 8 cases in which an esophagoscopy was done the roentgenographic diagnosis of hiatal hernia was uniformly confirmed, and strictures were observed, as were ulcerations in a few cases. The tissues removed revealed inflammatory changes of the mucosa. In certain other cases the mucosa was pale and smooth, and in one case there appeared a severe induration and thickening of the wall apparently due to a sclerodermatous process.

The symptoms of dysphagia were localized at either the upper or lower part of the esophagus, intermittent or constant, with pain and burning on swallowing as a rule.

The authors further state that relief or partial relief from these symptoms was obtained by various methods of dilatation of the esophagus, although recurrence at a later date was common. However, transient symptomatic relief only was obtained because the constitutional condition of acrosclerosis or scleroderma persisted and progressed. When these conditions are self-limited the esophageal lesions are not difficult to control.

It is important that the endoscopist recognize the association of esophageal pathology with the surface lesions of acrosclerosis and scleroderma in order to be able to assist the dermatologist in his treatment of these conditions and afford the patient temporary relief of symptoms.

McMAHON.

Two Cases of Mediastinitis Caused by Perforation of the Esophagus Treated by Penicillin, with Recovery.

Grez, Anibal: Revis. de Otorrlaring. 5:115 (Sept.) 1945.

This article deals with two cases of mediastinitis caused by perforation of the esophagus and outlines the author's procedure in the treatment of these cases.

Whatever excellent results can be achieved by exploration with the esophagoscope, the technique is delicate and should only be performed by one who has acquired necessary skill.

The most serious complication is mediastinitis caused by perforation of the esophagus, which may occur during exploration or subsequently as a result of traumatism produced by manipulation. During the examination perforation may be caused by the removal of foreign bodies with sharp edges and acute angles, forcing the scope through strictures or taking too much tissue for biopsy. Following the exploration perforation may occur because of injury inflicted by too much dilatation of a stricture or by forcing the scope blindly, both of which produce ulceration and necrosis of the esophageal wall.

Spontaneous perforations are produced by sharp-edged bodies of small size which penetrate the esophagus or cause an abscess which terminates in a mediastinal fistula.

The symptoms vary with the location of the injury. In the cervical region these lesions will cause pain at the base of the neck radiating to the shoulders and edema of the neck. In the thoracic region a lesion of the upper portion will cause interscapular pain and superficial respiration, while in the lower portion the pain will be referred to the epigastrium. In both instances the serious nature of the condition will be shown also by tachycardia, pallor and elevation of temperature.

Treatment consists of absolute rest to the esophagus, gastrostomy if necessary for nutrition, full doses of penicillin, maintainance of fluid balance by clysis and transfusion when indicated. A surgical consultant must be called when there is suspicion of suppurative mediastinitis.

HIGBEE.

MISCELLANEOUS

The Child's Acquisition of Speech.

Sheridan, Mary D.: Brit. M. J. No. 4402 (May 19) 1945.

Analyzing the data accumulated over a period of three and one-half years in studying the acquisition of speech of 650 normal children, the writer was impressed with the discovery of a definite pattern of speech development. The child's first responsive sounds to the speech and gestures of his attendants begin about the age of 8 to 9 months and are of the repetitive variety ("mum-mum-mum"). Soon the child begins to echo the cadences of adult speech and his babblings assume normal vocal inflections or tunes, at first with exaggeration of the loudness and shrillness of the voice. The first "words" or sound groups often consist only of vowels and a tune accompanied by explanatory gestures ("a-aw"!—that's all) or the word consists of a labial consonant, a vowel and a tune ("baw"—ball). The vowels appear sometimes before the diphthongs. The latter cause the child considerable confusion at first, so that the less prominent vowel sound of the diphthong is often omitted, or the two vowels are interchanged (lady—"leddy"; cow—"coo"; coal—"cowl").

The consonants tack themselves on as the vowels are being acquired, first at the beginning of the word and much later at the end and in the middle of the word. The first consonants acquired are usually the nasal sounds ("m" and "n") and the labials ("b" and "p"), followed by the "front stops" ("t' and "d") and the "back stops" ("k" and "g"). The sounds of "r," "l," "w," "y" are usually

late in appearing and are frequently interchanged so that a "rabbit" may be a "wabbit" or a "yabbit." The last single consonants to be acquired are the "h," "z," "v," "th," and their unvoiced equivalents, "s," "sh," "f." Of all the sounds the unvoiced "th" is the last to be fully appreciated. The double consonants are at first reduced to one single sound (train—"tain", spoon—"poon") and later both may be sounded but often with the use of the child's habitual substitutions (three—"fwee", drink—"dyink"). Dr. Sheridan is convinced that these substitutions are not due to muscular incoordination but rather to auditory confusions. She also feels that these consistent patterns of speech occurring in the normal course of development are not the result of some structural immaturity in the auditory nerve or its association areas, but rather the result of a psychological immaturity.

In the author's experience baby speech most commonly occurs in the neglected or isolated child, the spoiled only child, and in the over-indulged child. The best remedy for all these children evidencing "environmental" retardation is daily attendance at a nursery school.

SCHALL.

A Study of the Inheritance of Respiratory Allergies.

Stiles, Karl A., and Johnston, Elizabeth J.: J. Allergy, January, 1946.

The authors of this paper call attention to the reports of Clarke, Donnally and Coca, published in 1928, in which they showed that there was a tendency for children of asthmatic parents to develop asthma and those of hay fever parents to develop hay fever. However, it is true that progeny do not necessarily develop the same allergic manifestations as do their parents.

It was attempted in 1931 to show that transmission of hypersensitiveness from the mother to the child was carried through the placenta. In 1934, Bray showed that active sensitization in utero was possible in man, but that passive sensitization in all probability was rare. Attention is called to the fact that Bucher and Keeler reported allergy transmission as a dominant gene in five generations of 454 individuals.

The exact mode of inheritance of atopy is extremely difficult to determine because there are so many unknown factors.

One of the most interesting facts that has been brought out by Bray is that twice as many males as females show an inheritance of an allergic tendency before puberty, after which the clinical symptoms

of atopy appeared more often in females. He, therefore, concluded that puberty was a definite milestone after which a great number of males improved and greater number of females developed symptoms.

One of the things that these authors conclude is that the specific sensitivity does not appear to be inherited but there is inherited a capacity or predisposition to become hypersensitive.

McLAURIN.

Spontaneous Mediastinal and Subcutaneous Emphysema Complicating Bronchial Asthma.

Schwartz, Emanuel: J. Allergy, November, 1945.

In this paper, Dr. Schwartz is reporting one additional case of spontaneous mediastinal and subcutaneous emphysema complicating bronchial asthma, but he analyses 25 cases that have been previously reported. He calls attention to the essay of Macklin and Macklin entitled "Pulmonic Interstitial Emphysema" that included mediastinal emphysema, or pneumomediastinum, and subcutaneous emphysema. These two men showed that by their experiments the conditions mentioned were the result of an initial pulmonary interstitial emphysema and that the essential factor in its production is a pressure gradient between air in the alveolus and the connective tissue underneath that lead to a rupture of the base of the "marginal" type of alveolus. This made it possible for the air to seep through multiple ruptures in the alveolar bases so that it entered the pulmonic interstitial tissue. In this phase, the emphysema is seldom recognized clinically. The pulmonic vessels underlying the alveolar bases become compressed and there is a stagnation in the arterial and venous circulation. The air then travels along the vascular sheaths to enter the mediastinum. After the mediastinum becomes distended, there is sufficient pressure on the heart and the great vessels to create further embarrassment in the circulation. It is possible that some of the air may travel along the interstitial bands from the lung to the pleura, thereby forming a subpleural bleb which may rupture. However, this has not been demonstrated definitely. Before the Macklins made this study, it was thought that air found in the pleural cavity was the result of a rupture of an emphysematous bleb associated with minute tears produced by pull on adhesions, where spreading from the mediastinum could then produce a pneumoprecordium or an interstitial emphysema of the opposite lung or it could even enter the retroperitoneum. It may extend along the carotid sheaths and enter the subcutaneous tissues of the neck and head.

Spontaneous emphysema is far more prevalent than is supposed. One of the most interesting facts that Schwartz brings out in his paper is that although some of these patients may become practically moribund, they can show a very sudden improvement with lessening of the cyanosis, easier breathing and final recovery. The one finding that usually precedes such improvement is the appearance of the subcutaneous emphysema in the neck and face.

Dr. Schwartz also calls attention to the "crunching" sound heard over the heart in systole, associated with substernal pain and roentgen evidence of air in the mediastinum in cases of pneumoprecordium. The x-ray picture of the chest should be taken in a lateral position to show this air in front of the heart.

Another important observation is that subcutaneous emphysema is a very rare complication in bronchial asthma.

In Dr. Schwartz' case, no improvement occurred until the emphysema developed in the neck. All these cases that have recovered have cleared under rather conservative treatment, but it is highly necessary that oxygen be given to relieve the oxygen hunger and also to promote a more rapid absorption of the nitrogen in the tissues. Coughing should be controlled by sedatives and the barbiturates are definitely indicated. Adrenalin and aminophylline are also of considerable use.

McLAURIN.

Anaphylactic Shock From Skin Testing.

Swineford, Oscar, Jr.: J. Allergy, January, 1946.

Dr. Swineford reports two cases of anaphylactic shock, one of which proved to be fatal, following the routine plan of skin testing. He calls attention to the fact that other authors have reported deaths from anaphylactic shock and that there was one recently reported in which 10 grains of aspirin were taken by the patient.

In both of these cases that Dr. Swineford has reported, the skin testing was done by intradermal injection, and after they had proven negative to 72 of the commonly reacting allergens he did intracutaneous testing for 56 food extracts. He ran into trouble with these patients after eight or nine intracutaneous injections of inhalant extracts. One of the patients died in about ten minutes in spite of all efforts with vasoconstrictors and artificial respiration. The other patient survived after such measures but the reaction in that case was very serious.

The one very important conclusion that was reached by the author was that "every intradermal test should be preceded by the less sensitive scratch test." In both of the cases reported, the anaphylactic shock occurred before sufficient time had elapsed for any reaction to prove positive at the site of intradermal injection.

McLAURIN.

Relief of Dermographism and Other Urticarias of Histamine Origin by a Synthetic Benzhydryl Alkamine Ether.

Feinberg, Samuel M., and Friedlaender, Sidney: J. Allergy, November, 1945.

These two authors have called attention to the early reports of Lewis which indicated that urticaria factitia produced by a single stroke on the skin constituted an exaggerated physiologic response. Lewis and Grant also showed that stroking of the skin to produce dermographism created wheals that were practically identical to those caused by histamine in nondermographic subjects. These men showed that there was a general rise in skin temperature and flushing of the face following the extensive stroking of the skin of the trunk in subjects with urticaria factitia.

The principal reason that these two authors presented this paper was to call attention to the effect of a new synthetic chemical, B-dimethylaminoethyl benzhydryl ether hydrochloride, which is a definite antagonist to histamine and it also has the advantage of low toxicity. Antagonists to histamine previously reported have usually been very ineffective. This benzhydryl alkamine ether has been used in their out-clinic department to control a few cases of severe urticaria factitia of several years' standing. The drug can be taken by mouth and rather dramatic relief has been obtained from the whealing and itching even after the first dose was given. The dosage is from 50 mg. to 100 mg. three times daily, the patient adjusting the dose to his own requirements. One very valuable use of the drug was to make it possible in patients who are subject to dermographism to perform scratch tests and properly evaluate such tests. In these cases of dermographism, the amount of reaction from such scratch tests is very difficult to determine, but by the use of this drug, given to the patient in advance of the tests, the reactions to the scratches can be estimated rather accurately. They further have shown that the drug has no beneficial effect in the average case of rhinitis and asthma.

McLAURIN.

Books Received

The Etiology of Deaf-Mutism with Special Reference to Heredity.

By Harald Lindenov. Translated from Danish by Axel Anderson. Pp. 268. Illustrated with 176 family pedigrees and 34 audiograms. Copenhagen, Einar Munskgaard, 1945. (Price Dan. Cr. 25,00)

The introduction to this work includes a complete review of all recorded knowledge of deaf-mutism from the Middle Ages, in which Aristotle's erroneous conception of the nature of deaf-mutism prevailed, to all modern records in chronological order. Classifications by modern authors are presented, and the percentage of deaf-mutes per unit of population of all countries is quoted. All authors discussing hereditary deaf-mutism are reviewed, including data regarding marriage between deaf-mutes, consanguineous marriages, and the factors of recessive characteristics of deaf-mutism occurring in sporadic cases. Under the headings of acquired deaf-mutism causes are classed as constitutional disorders, trauma, acute and chronic infections, meningitis, intoxications, and otitis media.

Lindenov's investigation consists of personal examination of 32 families with deaf-mutes in a large group of island counties of Copenhagen and six other communities more isolated in location. The deaf-mutes in six national and city institutions are analyzed, 480 cases are reported in detail.

Remnants of hearing are found in a total of 50 per cent with an even distribution in different groups. Diminished vestibular irritability on one or both sides occurs in 12.1 per cent. Absence of the vestibular function caloric reaction on one or both sides occurs in 24.1 per cent. Ménière-like symptoms occur in 24.1 per cent of cases.

No clinical symptom is found that could be used as a means of differential diagnosis between hereditary and acquired deaf-mutism. The total number of deaf-mutes resulting from consanguineous marriages amounts to 6.3 per cent, while the frequency of such marriages in ordinary population is given as 2.1 per cent. In all pedigrees studied the predisposition to acquired deaf-mutism appears strongly probable. Retinitis pigmentosa is found in 58.3 per cent of cases, proving a strong correlation between retinitis pigmentosa and sporadic deaf-mutism. The high incidence of deaf-mutism as a result of any intermarriage of persons carrying the traits, is an emphatic indication for all eugenic restrictions from marriage.

COSTEN.

Diseases of the Nose, Throat, and Ear, Including Bronchoscopy and Esophagoscopy.

Edited by Chevalier Jackson, M.D., Sc.D., LL.D., F.A.C.S., Honorary Professor of Broncho-Esophagology, Temple University, Philadelphia, and Chevalier L. Jackson, M.D., M.Sc., F.A.C.S., Professor of Broncho-Esophagology, Temple University, Philadelphia, with the collaboration of 64 outstanding authorities. Pp. xiv + 844, with 934 illustrations on 581 figures, including 18 plates in color. Philadelphia, W. B. Saunders Company, 1945.

This latest contribution of the Jacksons is divided into eight parts. The first three pertain to the nose, mouth, pharynx and ear, the next four to the larynx and bronchoesophagology, while the last is devoted to general considerations.

The material is handled according to a definite format, each subject being discussed from the standpoint of anatomy, physiology, etiology and pathology, symptoms, diagnosis, complications, treatment, sequelae and prognosis. The separate phases of each subject receive the special consideration of different, well-qualified authors.

While the subject matter presented is accurate, it is not sufficiently complete, especially in the chapters on the nose and pharynx, to give the reader the exhaustive information that is a necessary prerequisite of a good textbook. But perhaps the authors did not intend that it should be regarded as such.

The chapter on the ear is well developed. The function of hearing, audiometric testing, and the value and limitations of the hearing aid are thoroughly discussed. Special emphasis is placed on otosclerosis, and the newer measures advocated for the relief of deafness due to otosclerosis are accurately described.

The chapters on broncho-esophagology are written with the usual Jackson attention to detail and clarity. Surgical procedures on the larynx, including arytenoidectomy for the relief of dyspnea resulting from bilateral abductor paralysis, are accurately and fully described. The remaining chapters are more or less a reiteration of their former publications which cannot be read too often, not only by those who are chiefly interested in broncho-esophagology but also by those who would acquire a fuller knowledge of otolaryngology.

The last chapter deals with aviation otolaryngology, chemotherapy, focal infection, surgery in tuberculosis and anesthesia.

While not a textbook per se, this volume should serve as a valuable source of reference for the otolaryngologist.

McMAHON.

Notices

AMERICAN BOARD OF OTOLARYNGOLOGY

The next examination of the American Board of Otolaryngology will be held in Chicago at the Palmer House from May 22 to May 25. Communications should be addressed to the Secretary, Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE

A one week didactic and clinical refresher course in Otolaryngology has been arranged for specialists in the field from May 13 to May 18, 1946, inclusive. In addition, a special course in Broncho-Esophagology will be given from June 3 to June 15, 1946, inclusive. It will consist of lectures, animal and cadaver demonstrations, diagnostic and surgical clinics. For information address Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago, Illinois.

UNIVERSITY OF IOWA

The State University of Iowa will offer during the 1946 Summer Session an intensive course in audiometry and the fitting of hearing aids. The course will run from June 17 to July 29 and is designed to train technicians as lay assistants to otologists, school nurses in public school hearing testing, executive secretaries in leagues for the hard of hearing, and others interested in hearing conservation. Any person who meets college entrance requirements is eligible. Applications may be made to Prof. Wendell Johnson, Director of the Speech Clinic, University of Iowa, Iowa City, Iowa.

OFFICERS

OF THE

NATIONAL OTOLARYNGOLOGICAL SOCIETIES

American Academy of Ophthalmology and Otolaryngology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
President-Elect: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5, Md.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Chicago, Ill., October 13-18, 1946.

American Broncho-Esophagological Association

President: Dr. Carlos E. Pitkin, Carnegie Medical Bldg., Cleveland, Ohio
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11, Ill.
Meeting: Drake Hotel, Chicago, Ill., May 31, 1946.

American Laryngological Association

President: Dr. Harold I. Lillie, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis 8, Mo.
Meeting: Drake Hotel, Chicago, Ill., May 29-30, 1946.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Albert C. Furstenberg, University Hospital, Ann Arbor, Mich.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Meeting: Drake Hotel, Chicago, Ill., May 27-28, 1946.

Sections:

Eastern—Chairman: Dr. Philip E. Meltzer, 20 Charlesgate W., Boston, Mass.
Southern—Chairman: Dr. E. Trible Gatewood, Professional Bldg., Richmond, Va.
Middle—Chairman: Dr. Lawrence R. Boies, Medical Arts Bldg., Minneapolis, Minn.
Western—Chairman: Dr. Clarence A. Veasey, Jr., 407 Riverside Dr., Spokane, Wash.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Secretary: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.
Meeting: San Francisco, Calif., July 1-5, 1946.

American Otological Society

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Gordon D. Hoople, Medical Arts Bldg., Syracuse 3, N. Y.
Meeting: Drake Hotel, Chicago, Ill., May 31-June 1, 1946.

First Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia, 3, Pa.
Meeting: Palmer House, Chicago, Ill., October 17-19, 1946.